Journal of Occupational Medicine

VOLUME II

January through December, 1960

OFFICIAL PUBLICATION OF

THE INDUSTRIAL MEDICAL ASSOCIATION

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blic Health

JANUARY 1960
VOL. 2 NO. 1

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Journal of Occupational Medicine

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Official Publication of the Industrial Medical Association

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Journal of Occupational Medicine is published by the Industrial Medical Association, 28 East Jackson Boulevard, Chicago 4, on the 1st of each month. Second class postage paid at Chicago, Illinois. Copyright 1960 by the Industrial Medical Association.

Subscriptions $10.00 per year—U. S. and foreign. Single copies $1.00 postpaid. Make checks payable to Journal of Occupational Medicine.

Change of address notices received by the end of any month will assure delivery of the following month's issue. Address change is facilitated if old address is also furnished.

Subscriptions, changes of address, and all business matters should be addressed to Clark D. Bridges, Publication Manager, Journal of Occupational Medicine, 28 East Jackson Boulevard, Chicago 4, Ill. Eastern Advertising Representative: Mrs. Virginia Page, 15 West 44th St., New York 36, N. Y. Telephone: Murray Hill 7-7040.

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Official Publication of the Industrial Medical Association

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The Industrial Medical Association was organized in 1915 as the American Association of Industrial Physicians and Surgeons. Its first annual meeting was held the following year. In 1951 the present name was adopted.

The purposes of the organization as stated in the certificate of incorporation (not-for-profit) are:

"The object of this Association will be to foster the study and discussion of the problems peculiar to industrial medicine and surgery; to develop methods adapted to the conservation of health among workers in the industries; to promote a more general understanding of the purposes of the medical care for employees; and to unite into one organization members of the medical profession specializing in industrial medicine and surgery for their mutual advancement in the practice of their profession."

Presently, the membership totals nearly 4000 physicians in the United States, Canada, Latin America, and other parts of the world. Physicians "who have an interest in Industrial Medicine" may apply for membership; specialization in this field is not essential. Regionally, the Industrial Medical Association has 14 Component Societies in the United States. Most Association members carry their membership through one of the component groups.

The Journal of Occupational Medicine, owned and published by the Industrial Medical Association, is sent monthly to each member as one of the advantages of membership.

The annual business meeting and convention is held in the spring. Since 1945, the convention has been held concurrently with the meetings of four other organizations having related interests—American Industrial Hygiene Association, American Conference of Governmental Industrial Hygienists, American Association of Industrial Nurses, Inc., American Association of Industrial Dentists. This combined meeting is known as the Industrial Health Conference.

The Occupational Health Institute, Inc., is a nonprofit educational organization created by the Industrial Medical Association to promote the cause of health in industry. Since 1951, the Occupational Health Institute has conducted the program of evaluation, approval and certification of Medical Services in Industry, on the basis of standards established by the Standards Committee of the Industrial Medical Association. This program was conducted by the American College of Surgeons for many years until its transfer to the Association.

The Association's headquarters office serves as a focal point for the Association's activities, and through its coordination of information regarding industrial health problems encountered throughout the country, it offers invaluable assistance to members on a host of specific subjects.

ANNUAL MEETING & INDUSTRIAL HEALTH CONFERENCE

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Journal of Occupational Medicine

This anniversary issue of the JOURNAL OF OCCUPATIONAL MEDICINE *presents articles by distinguished dermatologists and scientists who were invited to participate in a symposium on occupational dermatoses. The authors are from Great Britain and the United States, the two countries in which there has been a profound interest in occupational skin diseases; and they report research activities which range from those of physical chemistry to clinical dermatology. We hope that for the industrial physician, the dermatologist and the student of both fields, these papers individually and collectively will provide not only a source of new data and useful information, but a stimulus to explore the unexplored and reveal the ultimate simplicity or complexity of many of our cutaneous problems.—Editor*

Dermatology in Occupational Medicine

Raymond R. Suskind, M.D., Cincinnati, Ohio

Everything is simpler than you think and at the same time more complex than you imagine.
—Goethe

THE close relationship between occupational medicine and dermatology is an ancient one. It is as old as civilized man's awareness that illness may be provoked by his work or its environment. Reference is made by Celsus in the first century A.D. to ulcerations of the skin in workers who handled corrosives. Among the earliest occupational diseases described were diseases of the skin, and, since a cause-effect sequence was observed, these diseases were also among the first cutaneous problems for which specific etiologic agents were ascertained without difficulty. Industrial growth of the last 10 decades has given rise to many dermatological difficulties. These, like other medical problems, have been carefully described and classified.

Scope of Occupational Skin Diseases

During the more recent era of gigantic technological expansion the number of industrial processes involving physical and chemical exposures which may be injurious to the skin have increased. At the present time a large proportion of reported occupational illnesses are those which affect the skin. This is a logical reflection of the fact that the skin, with its large surface area in direct contact with the environment, is also the most vulnerable of organ systems. For these numerous problems of cutaneous damage both the industrial physician and the dermatologist have accepted the challenge of medical responsibility. This responsibility includes the development of new information regarding cause, pathogenesis, diagnosis, management, and prevention and the effective utilization of such knowledge.

The range of problems which faces the physician is enormous. A panorama of present-day occupational skin hazards might include types of industries as well as exposures to specific materials such as the complex and numerous operations found in the manufacturing of machinery; in the making of electrical and electronic equipment; in automotive and aircraft fabrication. It would include the gigantic chemical industry which has revolutionized industrial processes as well as everyman's life. The new cutaneous hazards associated with manufacturing, processing and/or the use of chemicals range from simple inorganic substances to

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complex, high molecular weight polymers. Conspicuous among the areas of potential hazard are petroleum distillates, synthetic organic solvents, plastics, rubber, complex organic substances used in explosives, photographic materials, agricultural chemicals, drugs and metals. Cutaneous hazards are found in the production and use of consumer goods such as textiles, their dyes or finishes, and in food processing. A numerically important group of skin hazards which persist are those which involve exposure to irritating acids, alkalis, wet work and cleansing agents. The physical agents which are part of this panorama include the sources of ultraviolet and ionizing radiation.

Interdisciplinary Coordination

New information regarding pathogenesis, diagnosis, treatment, and prevention is more often, as in other occupational diseases, developed by coordinated interdisciplinary activity and involves not only the probing dermatologist and industrial physician, but the skills and tools of the industrial hygienist, engineer, biochemist, physiologist, pharmacologist, and epidemiologist. The individual explorer in this field whose quest is that of new knowledge about a specific problem is usually one who, by education and experience, is conversant with the useful implements of several scientific disciplines, some of which he will employ appropriately in research activities and others in clinical management. Interdisciplinary coordination is well demonstrated in the papers which appear in this special issue devoted to dermatology. They represent research efforts in which the investigators, in order to study a problem effectively, used the experience, scientific concepts, clinical and investigative tools of several branches of knowledge, including their own.

Some Fundamental Requirements

Occupational skin diseases are most intriguing because through them we gain insight into the aberrations of cutaneous structure, cytology, metabolism, physiology, and chemistry as provoked by environmental stimuli. Although this is, admittedly, a limited area of dermatology, it provides a most revealing microcosm of the entire field. Environmental stimuli may, for example, disturb either structurally or functionally one or more of the numerous constituent parts of this heterogeneous organ system. In order to study such disturbances, the explorer must be conversant with the intact tissues, their normal dynamics and function, and the patterns of their reactions to all manner of exogenous stimuli.

This involves the acquisition of information regarding the nature of percutaneous absorption, the anatomical and physicochemical barriers, the surface defenses against chemical insult, and the defenses against infectious agents. It embraces a knowledge of the dynamics of the epidermis and its reaction to physical and chemical assault. It encompasses an appreciation of vascular responses of the skin, inflammation, and cutaneous sensation. It presumes the recognition and familiarity with the complex information concerning the immunology, biochemistry, and cytology of the hypersensitive state. It includes an understanding of reactions of the sebaceous glands to endogenous and exogenous stimuli, of hair development, growth, damage, and loss. It involves a knowledge of the biochemical synthesis of melanin, of pigment darkening. It embraces the effect of physical, chemical, and nervous stimuli on the sweat apparatus, as well as the role of sweat apparatus in water and mineral metabolism and temperature regulation, the provocation of granulomatous reactions by certain metals, and the response of the skin to exogenous carcinogenic agents and their accelerators. In attempting to find an explanation of occupational skin problems we come face to face with the fact that, although knowledge of normal skin physiology and biochemistry is developing rapidly, there are still large gaps which must be filled before we can understand the abnormal effects of environmental exposures. Although the resources for acquiring such knowledge are increasing, our present fund of information regarding the mechanisms of cutaneous response to occupational stimuli is still meager. In addition, the sum total of our understanding of many important environmental skin problems is somewhat restricted by the youthfulness of the particular medical interest and by the limited number of explorers who are willing to accept the challenge.

The Industrial Physician and the Clinical Problem

Those who are entrusted with the diagnosis and treatment of cutaneous diseases in industry are somewhat fortunate in that there is a considerable store of useful information and experience from which to draw. The large majority of occupational skin diseases are common, easily recognized problems which respond to relatively uncomplicated medications and hygienic procedures. Despite the wide range of

possible skin reactions to occupational environments, most of the problems stem from primary irritant sources or wet work and less frequently from allergic sensitizers. The frequency of photodynamic reactions, pilosebaceous, pigment, sweat gland, granulomatous, keratogenic, and carcinogenic problems is relatively limited, depending naturally on the types of cutaneous exposure in a particular plant or industrial area. The industrial practitioner, in order to manage dermatological problems effectively, must acquire the necessary knowledge and simple skills. For the unusual and difficult-to-manage problem he can consult the specialist. With experience he will learn that, if the diagnosis is correct, the therapeutic measures are straightforward and uncomplicated. He will also learn that among the major pitfalls to be avoided are iatrogenic damage by improper medication and failure to provide or use proper hygienic measures so that the inciting agent may be avoided or exposure limited. He will be a more effective physician if he has a working knowledge of the emotional and social factors which may influence the development of, the chronicity, and the sequelae of occupational skin reactions. He should be able to provide supportive care and help to relieve some of the stresses which produce anxiety.

Prevention and Control

Occupational dermatoses are in the main preventable diseases. In the realm of dermatology there are few problems which respond so easily to preventive methods as do the occupational dermatoses. Dermatological experience has shown that persons with certain skin diseases, such as atopic dermatitis, seborrheic dermatitis, and prior-existing eczematous dermatoses, are more vulnerable to specific work exposures in which there are irritants or sensitizers. Nonoccupational skin diseases may be aggravated by such exposures as well. Hence, pre-placement examinations and assignment of personnel to jobs in which the aberrant skin is not likely to react adversely is an important means of control.

The keystone to prevention, however, is a thoroughgoing knowledge of the biological and more specifically the dermatological effects of industrial materials and production procedures, a familiarity with conditions of work, and the technics by which exposure to hazard may be controlled. Where cutaneous hazards are known, the physician has at his disposal two general measures of prevention: (1) engineering devices, such as ventilation, automation, and closed process systems; and (2) measures which may be used by the worker himself, such as personal hygienic technics, e.g., cleansing agents, protective clothing, gloves, and barrier devices. These measures are effective only if there is close cooperation between medical and industrial hygiene personnel, who bear the responsibility for preventing skin diseases in the plant, and production as well as administrative personnel, who can put them into effect. The success of these measures depends to a large degree on good communication with all levels of employees who are involved in the hazard. While technical competence and knowledge constitute the foundation components of a control program, its effectiveness is directly related to the plant education program, interdepartmental cooperation, and continued guidance, inspection, and watchfulness on the part of the medical department.

Research

Study of the Clinical Incident

Where dermatological problems arise in an industrial environment, it is necessary not only to employ methods of clinical investigation in which the disease manifestations and their development are studied, but also to identify the variety of factors within the work environment itself—physical, toxic, and even emotional—which can provoke such disease processes. Here again coordination of effort is vital to medical accomplishment, and the research may properly involve: the industrial physician himself, who assumes initial responsibility for the problem; the dermatologist, who on the basis of knowledge and skill will determine the nature of the cutaneous reaction and what avenues of study should be followed; the industrial hygienist, who examines the work environment; the analytical chemist or engineer, who quantitates the intensity of the hazard; and the toxicologist, who develops knowledge regarding the biological effect. This type of probing attempts to answer questions regarding cause and control of a specific incident.

Epidemiological Investigation

Although the numerical incidence of occupational dermatoses as a whole is great, our statistical knowledge about the occurrence of problems of specific origin in relation to particular industry, or in relation to a regional or national population, is very meager. This is true for occurrence of such common problems as dermatoses due to machine tool coolants, insoluble cutting oils, plastics, heavy metals, non-

polar and polar organic solvents, and other specific classes of chemical materials for which information is more easily isolable. The statistics now being collected by state and national compensation or labor agencies in this regard are useful, but since reporting and classification are not uniform, the data from state collecting agencies are not comparable. Until we have planned epidemiological investigations of proper experimental design to determine *how much* we have of *what* problem and *where* it is found, we cannot continue to talk rationally about the progress made on a national scale to cut down the incidence of occupational dermatoses. We can, however, point to specific plants in which control programs and good epidemiological follow-up have provided considerable evidence of change. Unfortunately, too many of these figures remain unpublished. Furthermore, the problems which find their way into investigations by university, private, and government agency study units usually affect relatively large plants. The greatest segment of reported industrial skin problems still occurs in small plants where hygienic technics are inadequate, medical consultation limited, and research economically not feasible.

New Hygienic Methods

Although the methodology of occupational dermatoses control is fairly well developed, there still is considerable need for more effective technics. This is particularly true in the case of skin-cleansing products, neutralizers of specific chemicals, decolorizing agents, and protective creams, to mention just a few. It is not possible to touch upon this subject and fail to mention high-pressure promotion of certain improperly formulated and inadequately tested cleansing agents and protective creams. In such instances, the gullible industrial consumer has been literally duped into using the products on the basis of extravagant claims, only to find them ineffective and occasionally even hazardous. Here again there is an apparent need for counseling by the dermatologist regarding the relative merits of specific products. There is also a need for industry to support collectively research programs for the development of more effective technics.

Basic Research

Although most occupational dermatoses yield to medical therapy, there are a large number of very intriguing problems which, if they were better understood in terms of physicochemical, biochemical, physiological, and biological mechanisms, would cast considerable light on many nonoccupational aberrations of the skin. There is already a background of tradition in this connection. For example, the investigation of the cases of hair loss from exposure to chloroprene dimers in neoprene manufacture provided a stimulus for the further investigation of the role of other sulfhydryl-inactivating substances, akin to the chloroprene dimers, on keratinization and hair growth. The industrial experience of widespread and severe sensitization in the dye industry by halogen-substituted nitrobenzene compounds led to the development of a research tool which has been used for the past 25 years in experiments exploring basic aspects of the hypersensitive state. This was the first group of compounds through which it was possible to correlate sensitizing potential and chemical configuration. A clinical interest in miliaria, which in many instances is an occupational disease, led not only to the classical research on the origin of prickly heat, but to a better understanding of the response of the sweat duct to various types of injury as well as to a clearer differentiation between the various types of heat exhaustion. The commonly observed pigment loss from contact with the rubber antioxidant, the monobenzyl ether of hydroquinone, has provided for further clarification of the biochemistry of melanin and the competitiveness of certain hydroquinones for the enzyme tyrosinase. The recognition of toxic reactions and effects on the skin of substances which are percutaneously absorbed and the urgency for developing such information about particular classes of compounds to be used in the household, industry, agriculture, and warfare has fired the enthusiasm of scientists of many disciplines. It has made them aware of the need to study the complex mechanisms of skin absorption and has led to the development of new sensitive technics which reveal the routes and rates of penetration as well as the physicochemical factors which influence them. Observations made in the past several years with the aid of new technics has already begun to alter our concepts about mechanisms of absorption.

The new and inadequately examined areas in this field which need systematic exploration are truly enormous. Those who do not fear adversity, unsurmountables, and initial disappointments can accept the challenge. Here are a few puzzling problems waiting to be explored: what are the biochemical changes which occur in skin damaged by organic solvents or by repeated wetting; what is the site of "antibody formation" and the site of reaction in the skin's response to eczematous sensitizers; why does

reactivity of the skin persist after sensitization to some chemicals and not to others; what are the mechanisms by which the polyreactor is provoked not only by the original incitant but by physical stimuli and emotional stress? The answers to the many questions about occupational dermatoses are in some instances *simpler than we think* but in most, much *more complex than we imagine.*

Education

In the coordinated activities of occupational medicine and dermatology, the education and training of medical personnel has a distinct and important function. At the centers of graduate training in occupational medicine, where the industrial physician may acquire the knowledge and skills of his complex specialty, proper instruction in dermatology should be an important part of the curriculum. At this time, a systematic presentation of the dermatological aspects of occupational health is being offered at only a few educational centers. Through such courses the trainee has an opportunity to become conversant with most of the important aspects of occupational skin diseases. In these curricula he also has the opportunity of viewing dermatology in its proper relationship to industrial medicine and industrial hygiene and the role it can play in both clinical and research areas. There is a proper place, too, for intensive postgraduate short courses in occupational skin problems. It has been the experience of the author that when organized properly such instruction fills a rather wide educational gap and is very useful to the industrial physician.

IT IS RATHER obvious, then, that the association of dermatology and occupational medicine has been and can be a most fruitful one. The skills and knowledge of cutaneous problems are of considerable importance to this rapidly maturing brand of medicine—occupational health. Cross fertilization is possible in many areas. The coordination of essential interests and integration of fundamental activities will continue to be indispensable to the growth of both fields.

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Percutaneous Absorption

Methods of Study and Factors Which Influence Absorption

Irvin H. Blank, Ph.D., Boston, Mass.

TOXIC *reactions* to certain chemicals may be observed in persons whose only contact with these chemicals has been cutaneous. Systemic reactions are much less frequent than cutaneous reactions. A cutaneous reaction may be the manifestation of a physical, chemical, physiological, or immunological alteration of the skin.

Since the normal intact skin is relatively impermeable to most chemicals, a *systemic reaction* can follow exclusively cutaneous contact only when the chemical agent is so highly toxic that even the small amount which penetrates the skin can elicit a reaction. Systemic reactions are more likely to result from inhalation or ingestion of toxic chemicals than from cutaneous exposure.

Cutaneous reactions are not uncommon among industrial workers. Mild reactions, such as dryness, which are probably only seldom thought of as "toxic reactions," may result from the physical removal of constituents of the skin by such occupational contact agents as organic solvents and aqueous solutions of detergents.[1, 2] A destructive chemical reaction can result from contact with corrosive agents such as alkalies or strong acids. Localized vasodilatation or vasoconstriction, wheal formation, and localized sweating are physiological reactions which occur only after the external contactant has penetrated into the skin and reached the blood vessels and sweat glands. The process of sensitization, which must necessarily precede any type of immunological reaction in the skin, can occur only if an external contactant penetrates through the skin and comes into contact with the reticuloendothelial system, where antibodies are formed.

From this analysis of the type of toxic reaction which may follow cutaneous contact with an industrial chemical, it is apparent that any examination of the mechanism involved in such a reaction should include a consideration of the permeability of the skin to the chemical. One must know whether the skin is permeable to the contacting chemical, how far into the skin this substance can penetrate, with which specific components of the skin it comes into contact, and whether or not it can be disseminated systemically without undergoing change. Specific information about the relative rates at which a substance penetrates the skin and is stored and/or excreted often clarifies one's understanding of the manner in which toxic reactions take place. Knowledge of the details of percutaneous absorption is a valuable aid to clear understanding of the development and causes of the systemic and cutaneous reactions which may follow cutaneous contact with industrial chemicals.

Methods for Studying Percutaneous Absorption

Percutaneous absorption has been studied in many ways, but not all the methods employed for this purpose lend themselves to quantitative measurement of the rate of penetration into the skin; few of them determine the avenues of penetration; rarely is the ultimate deposition of the penetrating substance accurately made clear. In a critical review of the methods specifically designed for studying permeability of the skin, one should question whether the techniques employed permit determination of both the number of molecules which penetrate a unit area of skin in a unit time and the ultimate destination of the molecules.

Most of the methods used for studying per-

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This study was supported in part by a Research Grant (RG-4760) from the Division of General Medical Sciences of the Department of Health, Education and Welfare, U. S. Public Health Service, National Institutes of Health.

cutaneous absorption fit into the following classification:

1. Use of "models" into which penetration takes place
2. Study of changes in the histology of the skin
3. Use of tracers
   a. Dyes
   b. Fluorescent compounds
   c. Radioactive elements
4. Measurement of physiological reactions
5. Analysis of tissues: skin, blood, urine, etc.
6. Measurement of the loss of penetrant from the cutaneous surface
7. Measurement of penetration into perfused skin.

Use of "Models" into Which Penetration Takes Place

To study the penetration of some substance into a model designed to simulate skin is relatively simple.[3] If a chemical in a given vehicle, such as petrolatum, is applied to the surface of a model, the amount of that chemical which is transferred from the vehicle into the model may be measured with a certain degree of accuracy. With use of a variety of vehicles, the rates of transfer of a given chemical into the model can be determined, but such a study does not necessarily provide data comparable to those which might be obtained if skin were used instead of the model. The chief disadvantage of this type of study is the difficulty encountered in attempting to construct a model which satisfactorily reproduces the structural and chemical properties of skin. Agar gels, which have frequently been used as models, are unlike skin both structurally and chemically. Data obtained from them should not, therefore, be construed as evidence of what takes place in the skin.

Study of Changes in the Histology of the Skin

Studies of changes in the histology of the skin following application of various substances to the cutaneous surface can yield valuable information about the effect of these substances on the skin or certain tissues in the skin. Destruction of cornified epithelium could be detected by this means;[4] stimulation as measured by density of mitotic figures, and alterations of collagenous tissues[5] could also be demonstrated. Histological and histochemical techniques must be followed with precision in this type of work, and interpretation of the sections must be thoughtful and critical. Methods of this kind yield information about the specific tissues affected by any penetrant. To design an experiment in which histologic evaluation can yield quantitative information about penetration is extremely difficult. Rates of penetration cannot be easily determined by these methods.

Use of Tracers

The movement of a penetrant from the surface of the skin has been followed by means of tracers, i.e., dyes[6] or fluorescent[7] or radioactive[8] chemicals which have been added to the penetrant. This method of study is always combined with other techniques—for example, with histological examination or chemical analysis of the tissue. Various types of tracers have been used for many years, but recently many new and improved techniques have become available. The tracer method presupposes that the tracer and the penetrant act similarly and remain together during penetration. Unfortunately, this may not always be the case. For instance, if a dye is added to a colorless fat, one must remember that the dye may leave the fat to which it was originally applied and become attached to another fat which was naturally present in the skin. When this occurs, the movement of the dye within the tissues and the movement of the applied fat are not necessarily identical.

In a variant of this method, penetrants may be used which are in themselves colored, fluorescent,[9] or radioactive[10] and therefore need no attached tracers. Colored and fluorescent materials, when used in this way, do not lend themselves to quantitative measurement. Penetrants which are in their own right radioactive, on the other hand, may be measured in microquantities with great accuracy. For work with these latter materials this type of tracer technique is the most accurate now available, provided one is certain that the radioisotope remains in the unchanged molecule of the penetrant.[11] The use of radioisotopes for quantitative studies is discussed later in more detail.

Radioisotopes have been very helpful in tracing pathways of penetration. Autoradiographic techniques for biological tissues have been discussed in detail by Boyd.[12] Such techniques can easily be applied to skin.

It must be remembered that if a section of skin is prepared for autoradiography at a given time following application of a radioactive substance to the cutaneous surface, the resulting autoradiogram will show the location of the radioactive substance at the time the section was prepared but will not necessarily show the pathway by which the penetrant reached that location. Only a series of autoradiograms taken

at successive time intervals indicate the route taken by the penetrant in traveling from the cutaneous surface to the location shown on the final section.

Measurement of Physiological Reactions

The penetration of a pharmacologically active substance can be studied by observing the reaction which follows its application to the cutaneous surface. Such manifestations as wheal formation[13] and vasodilation, for example, result from the penetration of histamine and nicotinic acid esters. By this technique it is possible to demonstrate whether or not a substance which is capable of causing a physiological reaction can penetrate into the skin and, if it does penetrate, to determine the time required for the reaction to develop. It is difficult, however, to use a physiologic reaction as a "yardstick" for the quantitative measurement of the amount of material which has penetrated because the extent of the reaction is not always directly proportional to the quantity of penetrant present.

Analysis of Tissues

Analysis of tissues is probably the method most commonly used in studying percutaneous absorption.[14] It is simple to obtain samples of blood or urine at successive time intervals after application of a chemical to the cutaneous surface and to determine the amount of that chemical in the blood or urine. Small specimens of skin, obtained by biopsy either singly or in series, may be analyzed for the penetrant.

When the amount of the penetrant present in these tissues can be determined, one has a means of quantitatively measuring penetration. Certain errors are, however, inherent in such a method. It is unlikely that all of the material which has penetrated through a given area of skin in the living animal will be present in the specific tissue submitted to analysis. Rarely would all of the penetrant be eliminated in the urine. The amount of penetrant in the blood at any one time is the resultant of the amount which enters the blood stream and the amount which leaves it. Since any chemical which reaches the upper dermis is likely to be carried away from the skin by the lymph or blood, the exact quantity which has penetrated cannot be determined by analysis of the skin. When a toxic reaction is being studied in a specific tissue, it may be essential to determine how much of the applied chemical has reached that tissue.[15] Such a measurement cannot, however, be interpreted as indicative of the total amount of penetration which has taken place.

Another source of error in this method may be the ability of the living organism to alter the penetrant before it has been quantitatively measured. When such alteration occurs, more of the chemical penetrates than can be retrieved by chemical analysis.

A modification of this method, i.e., "calibration" of the animal to be studied, increases the accuracy of results.[16] Suppose that the blood is to be analyzed as a measure of penetration, but that some of the material reaching the blood is known to be lost to other tissues. If the penetrant is administered slowly by the intravenous route and the level of this substance in the blood is determined at successive time intervals, a "calibration curve" can be constructed which relates the amount of penetrant introduced into the blood stream to the amount of that substance actually present in the blood. If, subsequently, the penetrant is applied to a given area of the skin of this animal and the blood level of this substance determined at successive intervals, the amount of penetrant reaching the blood via the percutaneous route can be determined from the "calibration curve" which has been constructed from data obtained after intravenous administration. With a little experimentation the rate of change in the blood level can be made quite similar for the two methods of administration. This is probably one of the best methods available for quantitative measurement of percutaneous absorption in the living animal.

If excised skin is used for studying percutaneous absorption, chemical analysis of the samples of skin can measure penetration accurately, since, of course, none of the penetrant can be transferred to other tissues by the blood. When nonhairy skin is used for such a study, it is possible to separate the epidermis and dermis and to analyze each of these tissues separately. Thus, one can learn not only that penetration has occurred, but also the location of the penetrant. A refinement of this method is to remove the stratum corneum by successive strippings with pressure-sensitive tape before separation of epidermis and dermis.[11] It is rarely possible to achieve entirely clean removal of the stratum corneum in this way, but the greater part of this layer can be removed with the tape. With this refinement one can remove that portion of the penetrant which has entered the stratum corneum, but has not passed through the major barrier at the base of the stratum corneum. This procedure makes it possible to determine more accurately the quantity of penetrant which has passed below the major barrier.

Each of these tissue analysis techniques can be made simpler as well as more accurate if the penetrant can be synthesized so as to incorporate a radioisotope into the molecule and if the molecule can be shown to remain unchanged during and subsequent to penetration.

Measurement of Loss of Penetrant from the Cutaneous Surface

After a known amount of some chemical has been held on the cutaneous surface for a definite period of time, if all the remaining chemical is completely removed and quantitatively analyzed the amount of that chemical which is lost can be said to have penetrated into the skin.[17] A number of errors are inherent in this method: Complete removal is difficult; that portion of the chemical which has penetrated into the stratum corneum only is not differentiated from the portion which has reached the deeper layer; the amount of material which penetrates is usually so small that information about penetration may be lost in the actual determination of the chemical.

If the chemical being studied by this method is radioactive, loss from the surface of the skin can be simply determined by measuring the loss of radioactivity as shown by direct counts on the cutaneous surface.[18] That portion of the chemical which penetrates and is carried away by the blood stream is removed from the field of the detector and is not counted. Here, too, errors inherent in the counting method may be of the same order of magnitude as the loss in counts due to penetration, so that data obtained in this way must be viewed critically.

Measurement of Penetration into Perfused Skin

It is possible to perfuse certain areas of the skin of experimental animals. If a chemical is placed on a circumscribed area of the perfused skin, the quantity of the chemical which reaches the perfusate is a measure of the amount of material which penetrates and reaches the blood stream.[19] The techniques involved in perfusing the skin are tedious, but if perfusion can be accomplished this method would appear to give a reasonably accurate measure of rate of penetration of the unaltered chemical into the blood stream.

Factors Influencing Absorption

As yet there is no well-accepted evidence that energy supplied by the living cells of the epidermis is used in promoting the transfer, against a concentration gradient, of any substance from the environment into the skin of nonaquatic animals. It would appear, therefore, that penetration into the skin is primarily a diffusion process. Since it is probable that this diffusion traverses a multiphase system, any substance which penetrates into the skin probably has to pass from one phase to another across interfacial boundaries. In addition to the multiple phases present in the skin, there are often properties of the penetrant itself or its vehicle which may affect absorption; e.g., the penetrant often reaches the cutaneous surface in solution or suspended in a vehicle which may be single or multiphase. It is evident therefore that transfer of matter from the environment into the skin and thence into other body tissues is a complicated physicochemical process.

Some of the major factors which influence the movement of molecules of a substance through a vehicle, out of a vehicle, into the skin, and through the skin are (*a*) solubility of the substance in the various phases of the skin and the vehicle, (*b*) concentration of the substance in the vehicle, (*c*) diffusivity of the substance in each of the various phases and (*d*) chemical reactivity of the substance with various constituents of the skin. The rate of penetration is, of course, a function also of (*a*) the thickness of the rate-limiting barrier within the skin and (*b*) the temperature. Under normal conditions, it is probable that for man each of these two latter factors is relatively constant. It is unlikely that there is much variation in the thickness of the major barrier, and the temperature of the cutaneous surface is quite constant for most areas of the body of man in a temperate climate. In the investigation of penetration into excised skin, however, temperature can be varied over a considerable range and be used as an important investigative tool.

It is not difficult to determine the solubility of a substance in any vehicle, but to determine the solubility of any substance in a given phase in the skin is impossible because the exact structure or chemical composition of the various phases in the skin is not yet known. It may be hypothesized that in the course of penetrating the skin a substance will have to pass through at least one lipide and at least one primarily aqueous phase. A molecule of a penetrating substance which is strongly lipophilic would tend to be retained in the lipide phase, while one which is strongly hydrophilic would be retained in the aqueous phase. In order to undergo transfer from lipide to aqueous phase and vice versa, a molecule should be both lipophilic

and hydrophilic, that is, should contain a similar number of nonpolar and polar groups. The relative solubility of a substance in two phases determines its partition coefficient; if the solubility is nearly the same in both the lipide and the aqueous phase, the partition coefficient will approximate unity.

If this hypothesis is correct, a substance with a partition coefficient of 1 should penetrate most efficiently. Treherne[20] studied the permeability of various nonelectrolytes through whole excised rabbit skin and found that the permeability constant increased as the ether/water partition coefficient increased from 10^{-5} to 1. However, since he did not study substances whose ether/water coefficients were greater than 1, his work does not show whether at higher partition coefficients the permeability constant again decreases. A study such as this, nevertheless, makes quite apparent the fact that relative solubility in various substances greatly affects permeability.

Not only the solubility of a penetrant in the various phases of the skin, but also its solubility in its vehicle determines its rate of penetration. In industry it is possible that the skin may come into contact with a toxic agent which is dissolved in different vehicles. The agent is likely to be released to the skin more rapidly from that vehicle in which it is less soluble. Therefore, from two solutions of equal concentration, more of the toxic agent is likely to penetrate into the skin from that vehicle in which the toxic agent is less soluble. Penetration of a soluble substance from any one vehicle is facilitated by increasing the concentration of the toxic agent in that vehicle.

It is easy to understand why the rate at which the molecules move within a vehicle (diffusivity) or in any phase in the skin will influence the rate of penetration. This does not, however, seem to be a major factor in the control of percutaneous absorption, because molecular volume is the major factor which controls diffusivity, and the molecular volume of many of the materials with which the toxicologist is concerned does not vary widely. At least, the range of molecular volumes is much smaller than the range of partition coefficients.

The factors which have been discussed so far have been primarily physicochemical. The chemical reactivity of a substance also influences its ability to penetrate the skin. If an agent reacts easily with certain skin constituents, it will be held by those constituents and its movement will stop. An agent which reacts with something in the cornified epithelium will be prevented from penetrating through the major barrier at the base of the stratum corneum. If it does penetrate the barrier, reaches the epidermal cells, and reacts with them, the penetrant will be prevented from further movement and from systemic distribution. In reacting with the epidermal cell, however, the penetrant may produce a toxic reaction in the epidermis.

Thus far, only penetration through intact normal skin has been considered. "Intactness" of the skin is one of the most important factors which prevents penetration of toxic agents. Any small, physical break in the structural continuity of the skin greatly decreases its protective power. The break need not be very deep into the skin; it need only be through the major barrier at the base of the stratum corneum. Such breaks could be numerous and still not visible to the naked eye.

The structure of the skin may also be altered by chemical reaction or by the removal of various constituents by solvent action. Either of these alterations in the skin may be produced by the penetrant or by the vehicle in which the penetrant is held. Even weak alkalies are quite destructive. Organic solvents often decrease the ability of the major barrier to prevent the penetration of many substances.

Discussion

The industrial physician will ask how a knowledge of methods for measuring percutaneous absorption and a knowledge of factors which influence absorption will aid him in protecting the health of the individuals for whom he is responsible. As indicated in the opening paragraphs of this paper, percutaneous absorption is the first step in most toxic reactions which follow cutaneous contact with industrial chemicals. At the present time, an understanding of the methods used to measure absorption is perhaps of more importance to the investigator than to the industrial clinician. When, however, specific knowledge concerning the *factors which influence absorption* can be obtained, this will be of inestimable value to the industrial physician. With it, he will be in a much better position to protect the health of the group under his care. Unfortunately, it is now possible only to make informed guesses about many conditions for which complete knowledge is needed.

Some working hypotheses can be formulated. One can expect those substances which are both lipide- and water-soluble to penetrate more rapidly than substances which are primarily lipide-soluble or water-soluble. Within the range of molecular weights under 250, the low molecu-

lar weight compounds will not necessarily penetrate more rapidly than those higher on the scale. The specific properties of a penetrant are more important than its molecular size. Chemicals which are weakly soluble in their vehicle may penetrate the skin more easily than substances which are strongly soluble and are consequently held firmly by the vehicle. On the other hand, increasing the concentration of any one substance in any given vehicle in which it is soluble will probably accelerate penetration.

The process by which a vehicle promotes or retards the penetration of a chemical is complex. Those vehicles which hold the thermodynamic activity of a chemical at a minimum are likely to hold penetration at a minimum also, unless the vehicle modifies the skin. A vehicle which removes lipides from the skin may accelerate the penetration of water-soluble substance, but this will not necessarily accelerate the penetration of oil-soluble substances. A hot and humid environment which causes sweating may likewise accelerate the penetration of water-soluble substances, but not of lipide-soluble substances. Mechanical breaks in the major barrier, possibly only of microscopic size, in an otherwise normal skin, may be the most important factor in permitting penetration into the deeper layers of the skin. From working hypotheses of this kind, some rules may emerge which will aid the industrial physician in preventing those toxic reactions which result from percutaneous absorption.

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Extraction by Sweat of Chromium from Chrome Tanned Leathers

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THIS REPORT is a continuation of previous investigations[1, 2, 3] dealing with the action of chromates on the skin. The purpose of this study was to determine the capacity of sweat to extract chromium from chrome-tanned leathers and to identify the chromium compound released. The mode of action of perspiration in the breakdown of chromium-tanned leather was reviewed by Roddy and Lollar.[4] Cross-sections made of the damaged leather showed that the chromium migrated from the fibers into the interspaces directly below the grain surface, causing a discoloration. The damaged areas from the shoes contained a considerable amount of lactate, a major constituent of human sweat. In order to reproduce the cumulative effect of perspiration in leather, these authors used a synthetic sweat[5] containing 5% sodium lactate with a pH of 7.5. In their experiments they duplicated all the effects observed in badly damaged shoes, including discoloration.

On the basis of this finding, Morris[6, 7] contended that the chromium salts dissolved in sweat would come into contact with the skin. He implicated basic chromium sulfate, a trivalent complex chromium salt, as the chemical sensitizer responsible for some cases of shoe leather dermatitis. Morris therefore advised patch-testing with basic chromium sulfate. Furthermore, he emphasized that "neither sodium chromate nor potassium dichromate is found in shoes, and therefore these are not the chemicals with which people should be tested when they are suspected of being allergic to the chromium in their shoes."[7] The present work reports experiments aimed at testing the validity of these concepts.

This investigation was supported by a grant (No. E2346) from the Division of Research Grants, United States Public Health Service.

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Experimental Procedures

Human perspiration was collected from volunteers in the steam room of a health club. Large quantities were pooled and stored in the refrigerator until required for the experiments. The following experiments were carried out:

(a) One square-centimeter samples of 40 specimens of shoe leather, both new and old, were immersed in 5 ml of human sweat. One-half milliliter aliquots of sweat were tested for the presence of chromium with diphenylcarbazide after incubation for 24, 48, and 96 hours, 1 week, 2 weeks, and 4 weeks. No positive reactions for chromium were observed. Direct testing of the leather specimens with diphenylcarbazide gave a positive reaction for chromium in 10 of the 40 leathers.

(b) To enhance the extraction of chromium, the following additional procedures were carried out on the 10 leather specimens that gave direct positive reactions for chromium with diphenylcarbazide: (1) constant shaking and (2) incubation at 37°C. After varying periods of time the samples were retested. None of the sweat samples tested gave a positive test for chromium with diphenylcarbazide.

(c) Samples, 1 by 4 cm in area, of the 10 leathers used in *(b)* were immersed in 20 ml of sweat. One-milliliter aliquots were examined for chromium spectrographically at 24, 48, and 96 hours, 1 week, 2 weeks, 3 weeks, and 4 weeks. Chromium was present in all samples of sweat (Table I). The rate of extraction of chromium compounds by sweat showed no consistent relation to the extraction time.

The relative quantities of trivalent and hexavalent chromium extracted in 4-week-old sweat samples were determined with a polarographic method. Hexavalent chromium was present in all samples, and trivalent chromium was present in 6 of the 10 sweat samples (Table II). By a special technique[8] (Appendix) the leather specimens were analyzed and relative amounts

Table I—Amounts of Chromium Extracted by Sweat from Leather

Leather Specimen	Chromium in Sweat) (μg/ml)						
	24 Hr	48 Hr	96 Hr	1 Wk	2 Wk	3 Wk	4 Wk
1	020	050	050	080	050	060	100
2	100	180	080	100	200	100	280
3	180	150	100	120	180	100	200
4	350	300	180	080	100	120	220
5	100	220	150	080	150	050	100
6	050	100	080	070	100	150	120
7	080	150	100	200	080	080	100
8	060	160	120	300	090	020	060
9	050	100	040	050	080	050	050
10	070	090	060	080	100	090	080

Table II—Amounts of Chromium in Sweat and Leather Determined Polarographically

Leather Specimen	Chromium in Sweat (μg/ml)		Chromium in Leather (μg/cm²)	
	Trivalent	Hexavalent	Trivalent	Hexavalent
1	000	085	0842	0111
2	103	134	1207	0276
3	089	021	0700	0160
4	000	139	0776	0127
5	285	043	0811	0174
6	000	125	0782	0126
7	021	118	0685	0145
8	251	027	2318	0521
9	000	171	2498	0108
10	153	125	0352	0089

of trivalent and hexavalent chromium determined (Table II). This assay, however, did not define the nature of the hexavalent and trivalent chromium compounds, and further investigation will be required to clarify this point.

Comment

These experiments prove that human sweat can extract chromium compounds from shoe leather and corroborate previous observations made with a synthetic sweat mixture.[4] The concentrations of the chromium extracted by human sweat were insufficient to be detected by the diphenylcarbazide test; however, all samples showed chromium by spectrographic analyses.

Trivalent and hexavalent chromium compounds were demonstrated in the sweat by polarographic studies. Hexavalent chromium was present in all tested samples, whereas 4 of the 10 samples showed no trivalent chromium. It has been demonstrated that the valence of the chromium determines its diffusibility[9]; the greater diffusibility may account for its presence in all samples.

These findings do not support the concept that there is neither chromate nor dichromate in shoe leather. It has been well documented that hexavalent chromium has the capacity to sensitize human skin, whereas there is little if any evidence that trivalent chromium is a sensitizing agent.[10] Therefore, if an allergic reaction should be caused from shoe leather, one must consider the hexavalent chromium compounds as the more likely offenders for several reasons: (1) Hexavalent chromium has a higher sensitizing index; (2) because of their more ready diffusibility, hexavalent chromium compounds are more easily extracted by sweat; (3) the concentration of hexavalent chromium, although low, is comparable to its concentration in cement, which is capable of inducing sensitization[11]; while there is no evidence of similar reactions with such low concentrations of trivalent chromium.

The important role of sweat in extracting chromium compounds from leather has practical implications. The ability of sweat to extract chromium compounds from leather, however, does not necessarily imply that this type of exposure is a frequent cause for so-called shoe leather dermatitis. In our surveys of workers exposed to chromium compounds in various industries, we found but one instance of a so-called shoe leather dermatitis among 28 workers with proved chromate hypersensitivity. Because this patient gave a positive reaction to potassium dichromate, it was assumed that the chromium from the shoe leather caused the foot dermatitis. Unfortunately, this patient was not tested with the rubber adhesives and the thermoplastic rubber materials in his shoes, although he displayed irritation from adhesives. In our experience, the most frequent cause of shoe leather dermatitis is sensitization to rubber adhesives and thermoplastic rubber materials, as reported by Shatin and Reisch,[12] Fischer,[13] and others.

One can but theorize to explain the release of solubilized hexavalent chromium in sweat from trivalent chromium bound to protein during tanning: *(a)* It is possible that hexavalent chromium remains from a two-bath tanning process when this method of tanning is used. Since most leathers are now tanned by the one-bath method, this theory would not apply to the majority of leathers tested. *(b)* It is possible that during the hide neutralization process some trivalent chromium is oxidized to hexavalent chromium by some readily reducible substance (acting as an oxidizing agent) in the leather proper or more probably added during the tanning process (trypsin?).

Although trivalent chromium is present in larger concentrations in the leather, it is bound in a complex form to the protein; hexavalent chromium, which is attached to the protein with weak and unstable bonds, should be more readily leached out, as demonstrated by the polarographic analyses. The influence of the valence of chromium upon its diffusibility has also been reported by Hueper and Payne[9] in their concept of chromium carcinogenesis.

Summary and Conclusions

1. The extraction of chromium from chrome-tanned leathers by human sweat was demonstrated spectrographically. The findings support the observations reported by leather chemists.

2. The chromium compounds extracted were both hexavalent and trivalent as shown by polarographic studies. These results do not support previous reports which contend that a trivalent chromium compound is the sensitizing agent responsible for some cases of shoe leather dermatitis and that hexavalent chromates are not found in shoe leather. Since hexavalent chromium compounds are leached out by sweat, they cannot be dismissed as possible contactants, especially since hexavalent chromium is a well-known sensitizer.

3. The nature of the hexavalent and trivalent compounds will be the subject of further investigation.

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Appendix

Preparation of Samples (Technique of S. Katz[8])
Extracts—A 5 ml aliquot was pipetted into a 25 ml volumetric flask. The solution was neutralized to litmus with dilute HCl and dilute NaOH. One ml of 0.25% Triton X-100 was added, and the solution was brought to volume with 2 M KCl. A polarogram was then determined on a 10 ml aliquot.
Leathers—A rectangular portion of leather was cut from the sample and its area was determined by measuring its dimensions to the nearest millimeter. The portion of leather was then steeped to 10 ml of 6 N KOH in an atmosphere of nitrogen for 24 hours. A 5 ml aliquot was then treated as above.

The preparation of the samples for polarographic investigation was carried out with extreme care so that there was no chance of oxidation or reduction of the sample. The leather samples were decomposed with concentrated alkali and protected from oxidation or reduction of the chromium with an atmosphere of nitrogen. Under these conditions, the amounts of hexavalent and trivalent chromium found polarographically represent the true amounts present in the sample.

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CEMENT DERMATITIS

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ALTHOUGH there has been an upsurge of interest in dermatitis from cement over the past decade, dermatologists and industrial physicians have been aware of its harmful effect on the skin for centuries. Bernadino Ramazzini wrote a classic book "De Morbis Artificia Diatriba" (Diseases of Tradesmen), which was first published in 1700, when he was professor of medicine at Modena, in Northern Italy. He noted that "lime makes the hands of bricklayers wrinkled and sometimes ulcerates them." This effect he said could occasionally be used therapeutically, because lime freed men from "the scab" by virtue of its alkalinity. One of the first books on the same subject to be published in England was by Charles Turner Thackrah, who has sometimes been designated the "Father of British Industrial Medicine" (Hellier 1956). His book, which was first published in 1831, had a comprehensive descriptive title: "The Effects of the Principal Arts, Trades and Professions, and of the Civic States and Habits of Living on Health and Longevity with Particular Reference to the Trades and Manufactures of Leeds: and Suggestions for the Removal of Many of the Agents Which Produce Disease and Shorten Life." Naturally enough, he was mostly concerned with the more serious industrial diseases, but he does refer to "agents injurious to the skin, such as lime in bricklayers, which produce cutaneous disease."

As long ago as 1821 Alibert in France recognized lime as the cause of a chronic disabling dermatosis which he called "mellitagra" (honey eruption). But modern cement manufacture and the birth of a cement industry as such is usually credited to Aspden in England about 1824; and since then dermatitis from cement has been described in almost every country. Martial (1908) called it "la gâle du ciment" and estimated that a third of the workers were affected; while Anstett (1925) preferred the less specific label "cementers' itch." Artom (1921) in Italy gave an account of "the eczema of Masons," and O'Donovan (1925) in England described "lime dermatitis." The latter author saw many men affected by cement while they were working on the tunneling for the London Underground Railway system. He drew attention to variations in the clinical picture, and reported patients presenting with "cheiropompholyx," lime holes, seborrheic eczema, exfoliative dermatitis and erythroderma, pustular follicular dermatitis, intertrigo, and perforation of the nasal septum. He considered excessive sweating and overdryness of the skin to be predisposing factors and recommended certain preventive measures. In cement factories, the finishers, packers, and binders who seal up the sacks, are most liable to dermatitis, according to Holtzmann. The condition was described in Russia in 1931 by Joffe and Berline (cited by White[1]) in men who carried bags of cement on their backs and shoulders without adequate protection.

Prosser White[1] stated that lime dermatoses are largely seasonal. The backs of the hands and finger webs are always affected, in contrast to the sparing of the palms and occasional involvement of the face. The finger nails become thickened and dystrophic. Furunculosis, fissures, exudation and crusting are frequent clinical findings. The hands feel dry, stiff, and swollen after work, and smart so much at night as to prevent sleep.

In the United States much attention was devoted to the health of workers in cement manufacture, but mostly in relation to the dust hazard and chest illnesses. In a survey by Russell[2] of 150 plants employing 42,000 men he found that 10% of the sickness was due to skin disease and 70% of this was diagnosed as furunculosis. Whether these lesions were "lime holes" or pyococcal infections is uncertain. Writing in 1941, Anderson felt that "cement

This study was sponsored and supported by a grant from the Medical Research Council. Much of the chemical work was undertaken by Dr. A. J. M. Johnston, whose death occurred in June 1959.

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dermatitis deserves more considerations from the dermatologist, cement manufacturer, and insurance carrier than it has previously received." Keil[3] remarked on the difficult diagnostic problems involved, and the importance of the medicolegal consequences. He also explored some of the aspects of patch testing with hydraulic cement, lime, and similar building materials without coming to firm conclusions as to the value of such tests.

There can be little doubt that dermatitis from cement has been a constant problem over the past few decades in most countries, including France,[4-7] Italy,[8-9] Spain,[10] England, Switzerland[11] and America.[12] Over the last 10 years interest in the subject has intensified, as a direct result of the appreciation by Jaeger and Pelloni[13] in Lausanne, Switzerland, of the importance of its more than coincidental association with chromate sensitivity. It is interesting that Jaeger himself had been interested in the problems of patch tests with cement nearly 20 years earlier (1931). Bonnevie[14] and Burckhardt[11c] had observed positive patch tests to potassium dichromate in some instances of cement dermatitis, although they had both attributed their finding to the wearing of chrome-tanned leather gloves, albeit on inadequate evidence. The association of cement dermatitis with chromate sensitivity has been amply confirmed in the past decade on numerous occasions in many different countries, and the majority of authors conclude that allergic eczematous hypersensitivity to chromate is the principal cause of the dermatitis. In spite of this, I feel that such an explanation is too facile, and is not entirely justified by the facts. Similar doubts are expressed by Denton et al.,[15] who suggested that the chromate sensitivity may initiate and maintain cement dermatitis, without being wholly responsible in itself.

Chrome Compounds in Portland Cement

It is likely that cement in some form or other has been used for more than two thousand years. The Romans knew that limestone or chalk marls on heating and produces a cement-like material which sets when mixed with water. Over the past hundred years the use of cement has steadily increased, and extensive studies have been made of its physical and chemical properties. Although the word cement can be used to indicate any binding or setting material, including glues, in the present paper its use is limited to Portland cement. This can be defined as an intimate mixture of lime, silica, alumina, and iron burnt to the point of incipient fusion and ground.[16] Its precise composition is not known and almost certainly varies in different countries and in different factories, depending on the raw materials used. The finished product is a finely ground powder, made up of particles less than 0.089 mm in diameter ("flour") and a small proportion of somewhat larger particles ("residue").

The raw materials from which British Portland cement is made are calcareous matter, in the form of chalk or limestone, and argillaceous material, such as clay or shale, with the addition of gypsum. Coal is used as a fuel, and, as will be shown later, the incorporation of its ash in the final product is of some importance. The clay is mixed with water in a washmill to form a slurry. After removal of any sand from the clay slurry, crushed chalk or limestone is added, and the mixture is milled progressively until a fine and intimately mixed cement slurry is achieved. This "wet process" is very widely used in Britain, but in North America and certain European countries a "dry process" is more frequently employed. In the British method wet cement slurry, after a period of storage in tanks, is passed through a kiln, in which the temperature, is usually about 1200° to 1400°C. The kiln itself is a slowly revolving steel cylinder lying at the angle of an inclined plane. It is lined with special refractory bricks and may be up to 500 feet in length. The heat is provided by coal, and its ash is mixed with the cement clinker, forming about 5% of the finished product. Cement clinker in the form of "nuggets" from the kiln is mixed with gypsum (to delay the setting time when water is added), and then progressively ground by ball, roller, or other types of mill to a fine powder, after which it is ready for storage in bulk or bags.

This brief account of cement manufacture has been given in order to focus attention on the several possible sources of chrome compounds in the final product. Apart from the raw materials (chalk, clay, gypsum, and water), secondary sources to be considered are the coal ash, the grinding mills, and the refractory bricks of the kiln. In a 300-ft.-long cylindrical kiln, about 60 ft. would be lined with special chromium-containing bricks. The bricks are changed when they are half worn down—i.e., after about 12 to 18 months of use. The amount of chrome from the brick material, (containing 8 to 10% of hexavalent chromate) which finds its way into cement is extremely variable, but the theoretical maximum has been calculated as no more than 27 ppm.[17] In some cement-grinding mills chrome steel is used in the form of rollers or balls, because of its

Table I—Determination of Chromate in Raw Materials and Intermediary Stages

Material	Chromate (Expressed as $K_2Cr_2O_7$), $\mu g\%$
Cement	900
Gypsum	0
Clinker	893
Coal Ash	945
Slurry	0
Chalk	0
Clay	0

greater hardness. The wear on these mills is considerable, and certainly chrome steel gives them a longer life. Miescher et al.[18] thought that these rollers were a likely source of chromate in cement. From information on the "consumption" of chrome steel in one factory, the theoretical maximum was estimated at 200 μg of metallic chromium per 100 gm of cement.[17] However, the cost of chrome steel is high and by no means all manufacturers use it.

Little or no attention had previously been paid to the coal ash in regard to its possible chrome content. But it has been included in a recent analysis of such compounds in cement by Johnston and Calnan.[17] The chemical technique they used for hexavalent chromate was a modification of the colorimetric method of Spier and Natzel[19] which is based on the reaction with diphenylcarbazide.[20] The violet red color obtained with chromate in acid solution is specific, since only vanadium gives a similar color, but the latter is unstable and quickly fades. Total chromium was satisfactorily estimated by the fusion of cement with sodium carbonate and oxidation with bromine. The fusion has to be carried out in a platinum crucible.

As can be seen from Table I, the starting materials—clay, chalk, slurry, and gypsum—do not contain any chromate. The cement clinker, coal ash, and finished cement all contain appreciable, and approximately equal, quantities of chromate. The concentration in the coal ash, however, is not sufficient to account for the total amount in the final product, since only about 5% of cement is made up of such ash. It is clear that much of the chromate in cement appears during its passage through the kiln.

Manufacturers of cement have for a long time been aware of the presence of minute traces of chromium in their product. More recently water-soluble hexavalent chromate has been identified in cements from many European countries.[19, 21-24] In the United States Denton, Keenan, and Birmingham[15] confirmed its presence in every one of seven samples of American cement produced in various factories. Johnston and Calnan[17] examined 24 different specimens of British Cement, and found that the chromate content varied from 1200 micrograms per cent to 0 (Table II).

Of the seven samples which were free of chromate, none was colored, or made sulfate-resisting by the addition of iron and sand. But there is no reason to believe that this observation is significant.

There may be considerable disparity between the total chromium content of the cement and the amount present as hexavalent chromate. Denton et al.[15] often found the difference to be more than 10-fold, and Johnston and Calnan's findings[17] on eight samples are similar, as shown in Table III.

It is noticeable that in the specimens OPC(5) and WPC, containing no hexavalent chromate, the total chromium content was very low. One should of course bear in mind the fact that total chromium estimations depend on

Table II—Determination of Chromate in Cement (Expressed as $\mu g\%$ $K_2Cr_2O_7$)

Sample	Chromate Content	Sample	Chromate Content	Sample	Chromate Content
RPC(1)	1200	CPC(1)	330	OPC(8)	60
SPC	1200	OPC(5)	330	WPC	0
OPC(1)	1005	CPC(2)	300	OPC(7)	0
OPC(2)	990	CPC(3)	277	OPC(8)	0
OPC(3)	750	HPC(1)	244	HPC(2)	0
RPC(2)	690	RPC(4)	222	HPC(3)	0
OPC(4)	690	OPC(6)	180	OPC(9)	0
RPC(3)	540	OPC(7)	120	RPC(5)	0

Types of Cement: OPC, Ordinary Portland Cement
extra CaO. CPC, Colored Portland Cement
Cement. SPC, Sulfate-resisting, addition of iron and sand
WPC, white cement, free from iron
RPC, Rapid hardening
HPC, Hydrophobic Portland

Table III—Total Chromium Compared with Chromate Content of Cement (Expressed as $\mu g\%$ $K_2Cr_2O_7$)

Sample	Total Chrome (expressed as $\mu g\%$)	Chromate (expressed as $\mu g\%$)
SPC	11,300	1,200
OPC(1)	9,760	1,155
OPC(2)	3,600	990
RPC	1,995	690
OPC(3)	1,965	180
OPC(4)	1,140	330
OPC(5)	820	0
WPC	60	0

OPC, Ordinary Portland cement
SPC, Sulfate-resisting
RPC, Rapid hardening
WPC, White cement

Table IV—Determination of Total Chromium in Raw Materials and Intermediate Stages (Expressed as μg% $K_2Cr_2O_7$)

Material	μg%
Cement	9,760
Gypsum	2,920
Clinker	9,760
Coal ash	32,160
Chalk	740
Clay	21,840

Table V—Determination of Chromate in Cement Materials Before and After Burning (Expressed as μg% $K_2Cr_2O_7$)

Material	Total Chromium Content	Hexavalent Chromate Before Burning	Hexavalent Chromate After Burning
Slurry	10,340	0	300
Clay (1 part) / Chalk (3 parts)	21,840 / 740	0	690

the assumption that all of it is oxidized to chromate by the method used—an assumption that may not always be justified, with consequent inaccuracies in the results.

In contrast to the absence of any hexavalent salt in the raw materials, appreciable amounts of chromium in other forms have been found in the clay, chalk, gypsum and coal ash (Table IV) by Johnston and Calnan.[17] One may reckon that 130 parts of cement are produced from 120 parts of chalk, 40 parts of clay, 5 parts of ash, and 5 parts of gypsum (about 40 parts of the total are lost in the process of manufacture). On this basis, using the data of Table IV, 70% of the chromium in cement would come from clay, 13% from coal ash, and 7% from chalk, leaving 11% from other sources.

It seems clear from the above data that a proportion of the divalent and trivalent chromium in the raw materials, especially the clay, is converted to the hexavalent form in the kiln. Such an oxidation procedure can, in fact, be duplicated in the laboratory, by making a wet clay and chalk slurry and heating it in a platinum crucible to 1400°C in a sufficiently aerated atmosphere.[17] Table V shows the amount of chromate that could be produced by this method.

Occupations

The number of persons whose skin is exposed to cement must be enormous. From the point of view of dermatitis, the men involved in its manufacture are almost the least important group. For, although Russell's survey[2] of 42,000 men in 150 cement plants showed skin disorders to be the second most frequent disability, their usual complaint was "furunculosis" and not dermatitis. In modern cement factories dermatitis is infrequent, in spite of the fact that cement dust is such a fine powder that it must be virtually impossible to control it completely. Apart from reduction of the hazard by mechanization and automation in the plant, men are here exposed to dry rather than wet cement, and hence avoid slaking of the lime. But almost any worker in the building trade is a potential victim. This group includes a large body of general unskilled laborers, as well as bricklayers, plasterers, concrete mixers, paperhangers, plumbers, painters, whitewashers, and electricians.

People other than manual workers may also be exposed in the building and construction industries, as is illustrated by the well-known case of the "tea-boy" described by O'Donovan (1925). This boy's occupation was to make tea several times a day for the workmen and to carry it to them on a building site. During these journeys a certain amount of cement dust inevitably got into his socks and boots, and he developed a dermatitis of the feet from it.

Some artists and others who work with plaster-of-Paris sometimes add a little Portland Cement to their material. A hospital artist, Marcel Boudouin[25] described his own dermatitis from this cause when he was making the casts for producing wax *moulages* in Paris.

In addition to the various trades and professions which involve the handling of cement and cement products, one should remember that, with the modern vogue of "Do it yourself," almost any one can be exposed while doing some amateur cementing or plastering job at home, whether it is a major construction or a small repair. For this reason detailed lists of occupations in which cement products are handled is not especially helpful. It is wise to consider the possibility of "cement" in almost every instance of contact dermatitis, especially when it is chronic, and to ask suitably searching questions. The use of the chromate patch test in this regard will be considered later.

Clinical Picture

Anyone who has worked with lime or cement is aware of the dryness and tightness of the skin of the hands which it produces. The skin may smart and burn to cause considerable discomfort and loss of sleep. Most of the patients afflicted with cement dermatitis pass through this phase, although subsequently they may undergo a period of "hardening" before anything more untoward occurs. At times dryness

of the skin of the hands may progress to desquamation and fissures, with perhaps some thickening, linear striations, or other dystrophy of the fingernails. The metamorphosis into a frank dermatitis may be either abrupt or insidious. The changes are most often subacute and chronic, but on occasions severe acute vesicular or bullous eczematous lesions with gross edema are seen. Prosser White[1] said that "the backs of the hands and between the fingers are always the seats of trouble, the face sometimes, the palms never." Freedom of the palms is undoubtedly a frequent finding, but I doubt if one can agree that it *never* occurs. In workers in specific occupations, such as masons or plasterers, certain parts of the hands and fingers may be particularly heavily exposed to wet cement and friction, and consequently such persons may show localized areas of dermatitis, especially in the early stages. Several of our patients have said that cement plaster constantly falls on one area of their hand or forearm, precisely at the spot where their eruption began.

Spread onto the wrists and up the forearms may be associated with dermatitis of the hands. It may be in the form of a diffuse scaly or papular erythema and may merge with flexural involvement at the elbows. Less often it is exudative.

Dermatitis of the feet, as a manifestation of damage from cement, is common. It may occur by itself or in association with eczema of the hands. It may not always be easy to decide whether the foot eruption is due to direct exposure to cement or is a secondary phenomenon, since one knows that an eczema of the hands from any cause, whether exogenous or endogenous, has a tendency to spread to the feet. From my own experience I favor the view that it is most often produced by cement dust getting into the footwear. The pedal eruption is usually a fairly diffuse dermatitis of the dorsa of the feet rather than the soles and spreads up round the ankles and the lower legs. It may be sharply demarcated at its upper limit by the shoes. It has been suggested that much dermatitis of the hands and feet in cases of cement dermatitis is brought about by allergic sensitivity to leather gloves and footwear, and such a view was believed to be supported by patch test reactions to potassium dichromate. The distribution, however, of the eruption in cement workers is rarely as precise and defined as that found in cases of footwear dermatitis (Calnan and Sarkany, 1959). A great deal of dermatitis of the hands and feet, irrespective of the etiological factors, is chronic and recalcitrant.[26] Cases attributed to cement are no exception. When venous stasis and gravitational factors are present, as is not infrequently the case in older men, the lower leg may be prominently affected, and the differentiation from a primary hypostatic dermatitis becomes difficult. Venous stasis will naturally retard recovery even after removal of the patient from occupational contact.

A number of patients exposed to cement and similar products develop an eruption indistinguishable from discoid or nummular eczema. Virtually nothing is known about the causation of nummular eczema, and one can only speculate about the role of cement, when it is present in such cases. A proportion of the patients are allergic to chromate, and this is often used as evidence for implicating cement as the cause. But the precise relationships between nummular eczema, chromates, and the occupation remain obscure.

A not uncommon clinical pattern found in cement workers is "seborrhoeic dermatitis"—presenting as flexural, intertriginous lesions, peteloid eruptions on the chest and back, and lesions over the face, scalp, neck, and ears. Again the appearances are often those of a constitutional eczema, but they appear to be more frequent in the occupational group under discussion. The majority of the patients are sensitive to chromate. The picture is especially seen in those men who are most exposed to cement dust, and whose clothing is heavily impregnated. Their work is often hot and heavy, and they sweat profusely. It is readily understandable that cement dust settles on skin areas which are sweating freely, and it will then be mixed in solution, resulting in some generation of heat and a rise in alkalinity, consequent upon slaking of the lime. Some men show a very striking involvement of sites such as the waist under a tight trouser belt from this mechanism. The groins and genitalia may also be affected.

Lastly, any one of the above clinical patterns of cement dermatitis may become generalized, and lose all trace of identifiable initial features. The patient is occasionally first seen therefore with an exfoliative dermatitis or erythroderma.

Patch Testing

There is fairly general agreement that any direct skin tests with cement are without value.[4] Neither positive nor negative results should be allowed to influence the diagnosis. Such tests give very variable and inconsistent results. A test may be positive at one site, and

not at another—there is no satisfactory evidence to support the concept of localized sensitivity—or a test may be positive at one time and negative at another. Any positive result from cement as such can, with some confidence, be regarded as a toxic, irritant, or false positive reaction. More often, however, tests with cement are negative, in spite of its self-evident irritant properties.

Bonnevie[14] in Copenhagen, in his monograph on the etiology and pathogenesis of eczema, described 29 men sensitive to chromate. Nine of them wore chrome-tanned leather gloves, and, of these, 4 were masons and 3 were cement workers. He related the chromate reaction to the wearing of gloves. Pirila and Kilpio,[27] in a paper on dermatoses from bichromates, found 10 positive reactors (33%) among 30 cement workers whom they tested. For some reason which is not entirely clear, Jaeger and Pelloni[13] obtained a much higher percentage than this, and, as a result, they really appreciated the significance of the chromate test. In routine patch testing of 200 patients with eczema, their results were as follows:

Diagnosis	Positive	Negative	Total
Cement eczema	30	2	32
Other eczemas	9	159	168

Their principal findings have been amply confirmed, although most investigators have been unable to match their very high proportion of positive reactors.[18, 19, 28–43] My own experience is that about two thirds of cement dermatitis patients react to chromate.

There has been some difference of opinion on the question of the concentrations most suitable for testing. The standard solution usually recommended is 0.5% of potassium dichromate in water.[44] This often produces an unnecessarily strong reaction, and, in fact, the majority of patients give a satisfactory response to 0.1%. One must admit, however, that occasional patients will react only at the high concentration. Likewise, a few subjects are sensitive to 0.01% or even 0.001% and less.

Denton et al.[15] tested a patient known to be exquisitely sensitive to chromate with the filtrate from cement washings suitably buffered to p.H 5.5 and obtained repeatedly positive reactions. The concentration of chromate in the filtrates from two samples were equivalent to 0.0001% and 0.0004% respectively. But few patients with cement dermatitis are sensitive to this degree.

We have found that all our patients who were patch-test-positive to 1:1000 chromate solution also responded with a delayed tuberculin type reaction to 1:100,000 solution when injected intradermally. Although Haxthausen[40] has recorded evidence that reactions do not always run parallel, we have not yet found any disparity between the patch and intradermal tests at these dilutions. Hence, we have no reason to believe that intradermal tests are especially indicated.

Jaeger and Pelloni[13] found that none of three patients tested showed any reaction to trivalent chromium. In a larger series we obtained the same negative results. There is, in fact, serious doubt as to whether trivalent chromium is a sensitizer at all. The only evidence to the contrary is the single patient described by Morris.[46]

Nature of Cement Dermatitis

Burckhardt[11c] denied the existence of a genuine cement eczema, a term which he presumably used to describe a true allergic sensitivity to any constituent of cement. He felt that the irritant effects of hydrated cement were directly attributable to the calcium and hydroxyl ions. In a series of 80 patients whom he investigated, he diagnosed 72 (90%) as having primary irritant or toxic dermatitis, and the other 8 as having seborrheic, infective, or endogenous eczema; and whenever he obtained a positive patch test with cement, he regarded it as a false reaction—a type of Koebner effect. One of his patients was chromate-positive, which he attributed to leather gloves. Keil[3] did not entirely agree with Burckhardt's views.

There are, of course, adequate physical factors in cement to account for its irritant properties. Although it is mostly a fine powder, there are some larger particles which can abrade the skin; and, when used for mortar, it is mixed with sand particles which are also abrasive. It is alkaline, and when mixed with water, the pH rises even higher. Furthermore, some heat is generated at the time. Cement is also moderately hygroscopic. All these factors, either separately or in combination, are capable of damaging the skin, and it would not appear necessary to invoke any other mechanism.

The close association between chromate sensitivity and cement dermatitis is now proved beyond any doubt. The problem which remains is to assess the precise role and importance of this finding. While many authors on the subject conclude that the cause of cement dermatitis is chromate sensitivity, others are not prepared to accept the relationship as so direct. Denton et al.[15] stated that "moistened cement undoubtedly exerts a primary irritant action on the skin; however, the recent findings of positive patch tests to hexavalent chromium in ce-

ment users indicates that allergic chromate sensitivity may initiate and maintain cement dermatitis. Soluble hexavalent chromium is apparently present in all cements to a varying degree. Cement artisans are exposed to higher hexavalent chromium levels than is indicated by analysis of cement washings, for, as moisture evaporates from cement slurries in contact with the skin, the percentage of hexavalent chromium rises."

Samitz[47] has made a particular study of chromate dermatitis, and was impressed by the chronicity of such cases, in spite of their apparent removal from contact with the causative agent. This is certainly true of cement dermatitis, which in our experience is commonly an extremely chronic and intractable condition. From spectrographic analysis of skin biopsy specimens, he could find no evidence of the persistence of chromium in the local area one week after application of a chromate solution as a patch test, even though the area was still showing objective signs of a positive reaction. This is in agreement with other studies, which indicate that a contact antigen soon disappears from the local area of application and is evidently not required to remain there for the development of a reaction in a sensitized subject. Samitz also showed, by means of a spot test using the diphenylcarbazide reaction, that traces of chromate can persist on towels, clothing, and similar articles even after they have been through a laundry more than once. A quantitative analysis of a 6-in.-square piece of used toweling from a printing works revealed 100 ppm of chromium. What Samitz did not show, however, was that the use of such towels contaminated by chromate was capable of irritating, exacerbating, or prolonging dermatitis. If he had produced evidence on this point, it might have been most valuable. In one of our patients with a diagnosis of cement dermatitis and having a positive patch test to chromate, we applied to one affected limb an ointment containing 1 ppm of potassium dichromate and an equivalent control ointment to the opposite limb. The chromate-containing ointment considerably aggravated the dermatitis, whereas the control ointment did not.

Burckhardt[48] and some other workers feel that diminished alkali neutralization power and alkali resistance play an important part in cement dermatitis, both directly and indirectly by predisposing the skin to allergic eczematous hypersensitivity to chromate. It even has been suggested that patients may give a positive patch test to chromate only on an area of skin which has been pretreated with alkali. This can undoubtedly happen, but the introduction of such additional factors into the patch testing procedure raises serious difficulties in interpreting the results. A positive reaction obtained by such a method may not necessarily be indicative of true allergic hypersensitivity.[49] Furthermore, the reliability and validity of tests for the alkali neutralization and resistance powers of the skin are not universally accepted; nor have they become routine procedures in many countries outside Switzerland.[50]

How can this enigma of the precise nature of cement dermatitis and its pathogenesis be resolved? A great deal more experimental work and clinical study are required. It is possible, if not probable, that the pre-1950 views are still largely correct and that the importance of chromate sensitivity has been grossly exaggerated. The chromate in cement may be acting purely as a secondary factor. The patients develop an irritant or toxic dermatitis from the cement, which later becomes complicated by a superimposed dermatitis venanata from sensitization to chromate. One may regard it as analgous to the contact dermatitis of the hands so frequently seen in women who handle alkalis, detergents, and similar cleansing agents: to avoid aggravation of their dermatitis they begin to wear rubber gloves, to which some of them will also become reactive. Their hand eruption, however, is primarily an irritant dermatitis, and often persists after they abandon the use of rubber gloves. Again, patients with any type of eczema may become sensitized to one of the medicaments they apply; but the dermatitis venanata is only a minor and secondary etiological factor. In our present state of ignorance such a view of cement dermatitis can be no more than speculative, but it is deserving of consideration.

Summary

The harmful effects of cement on the skin have been appreciated for centuries. The incidence of dermatitis is low in cement factories but comparatively high among the wide range of occupations employing wet cement. In Britain it is especially frequent among unskilled laborers employed in the building trade. Interest in the subject has been reawakened over the past decade because of its association with allergic hypersensitivity to hexavalent chromium salts.

Soluble chromate has been identified in most, but not all, of 24 samples of British cements. Chromium, but no chromate, has been found in the raw materials from which cement is manu-

factured. Several possible sources of chromate in the final product have been considered, such as chrome steel used for milling, refractory bricks in the kiln, and coal ash. Quantitative chemical studies indicate that most of it originates in the kiln, by oxidation of trivalent chromium present in the cement slurry. The process can be duplicated in the laboratory, and from this fact it is clear that conditions in the kiln are adequate for such an oxidative reaction.

The occupations of persons exposed to cement and cement products are very diverse; they include building construction, plumbing, bricklaying, plastering, and painting and decorating.

The clinical picture of dermatitis from cement may often simulate a nonoccupational eczema. The main patterns are dermatitis of the hands and forearms; dermatitis of the feet; nummular dermatitis; seborrheic eczema; stasis dermatitis; and, occasionally, exfoliative dermatitis. These patterns may be mingled. Chronicity, recurrences, and therapeutic recalcitrance are also prominent features of the condition.

Between 50% and 90% of cases show a positive patch test reaction to potassium dichromate, and the test is of some value diagnostically. A 0.1% concentration of this salt is usually satisfactory, although 0.001% is adequate for an intradermal test. Trivalent salts give no true allergic reactions.

The nature of cement dermatitis is discussed, particularly in relation to the role of the chromate hypersensitivity. Cement dermatitis is not a chromate dermatitis but is a primary irritant dermatitis, complicated, in the majority of instances, by a secondary contact sensitivity to hexavalent chromate.

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The "Ruster" in Industry

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THAT PERSON employed in industry who, on touching a freshly polished metal surface* with the palmar surface of his fingers, causes severe corrosion to develop on that metal surface, is called a "ruster." Rusting of metal by palmar sweat is a specific instance of atmospheric corrosion. The "ruster" presents a real problem in industries where corrosion of highly polished metal surfaces is harmful. This paper is concerned with determining the component or components present in the skin surface film which cause the reaction, and with the development of a rapid screening test for detecting the "ruster" in industry.

The whole concept of transfer of skin surface film to finished product and its subsequent production of deleterious effect is not new. Supervision concerned with inspection and quality control has long been aware of and concerned with this problem. The photographic industry has been particularly cognizant of the effect of skin surface film on sensitized products. Other industries, such as those manufacturing tools, ball bearings, watches, locks, and electronic equipment should also have considerable interest in the "ruster." To approach this problem in systematic fashion, it would seem reasonable (1) to review the constituents of the skin surface film, (2) to consider the mechanism of the actual transfer of this material to metal surfaces, (3) to study the reaction on the metal surfaces and the ambient weather conditions which influence the reaction, and (4) to consider any preventive techniques, including a screening test for detection of potential "rusters."

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*The mild steel used throughout this study was of the "cold rolled type" and gave the following chemical analysis: carbon 0.15-0.25%, manganese 0.3-0.9%, silicon 0.3% maximum, phosphorus 0.04% maximum, sulfur 0.05% maximum, copper and vanadium in slight traces, remainder iron.

The Constituents of Skin Surface Film

The skin surface film of man is made up of an emulsion (roughly 50% each) of an aqueous and a lipid phase. It is difficult to determine just how much the eccrine sweat glands contribute to the maintenance of the surface film. Suffice to say, that sensible perspiration (eccrine sweat), insensible perspiration, and moisture from the environment are available to contribute to the aqueous phase of the skin surface film. In the adult, the largest contribution to the lipid phase is made by the sebaceous glands. Keratinizing epidermis also carries a great deal of lipid substance to the skin surface as its invisible horny lamellae are cast off. Relatively minor amounts of lipid are added through secretions of the apocrine glands. Although there are no sebaceous glands in the skin of the palms or palmar surface of the fingers, sebum flowing from the dorsal aspect of the hands and fingers and that transferred when the palms touch other skin surfaces of the body, certainly add to that supplied by the keratinizing epidermis in providing an emulsified lipid film on the palms and palmar surface of the fingers.

Chemical analyses of skin surface film have revealed lipids made up of free and combined fatty acids (both long and short chains), wax alcohols, hydrocarbons, sterols (including cholesterol), triglycerides, and triterpenoid alcohols. Only traces of nitrogen and phosphorus were found to be associated with the lipids. In the hand which is not visibly sweating, the aqueous phase contains water-soluble eccrine sweat constituents and nitrogen from keratin and from insensible perspiration. Chemical analyses of eccrine sweat under normal circumstances have revealed urea, creatine, creatinine, uric acid, amino acids, choline, glucose, lactic acid, traces of water-soluble vitamins B and

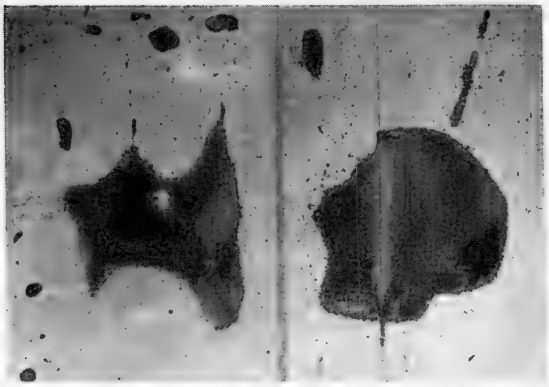

Fig. 1—Polished mild steel surface showing corrosion. Area on left (1) resulted from application of two drops of a 0.5% by weight sodium chloride solution—0.85% solution was applied to area (2) on the right. Photographs taken some three months after application of chloride.

C, sodium, chloride, potassium, calcium, magnesium, iron, sulfates, and phosphates. Palmar sweat which is secreted intermittently would undoubtedly influence, at least temporarily, the aqueous film on the palms because of its much higher concentration of electrolytes and organic material. For a more detailed discussion on the composition of the skin surface film, the reader is referred to Rothman's work on the physiology and biochemistry of the skin.[1]

The Rust-Producing Factor in Palmar Sweat

It has been demonstrated experimentally by Collins,[2] and earlier British workers, that high concentrations of *chloride* ion in sweat is primarily responsible for its adverse effect on metal surfaces. Fig. 1 demonstrates the corrosive action of 0.5% and 0.85% aqueous sodium chloride solutions on mild steel.

Spectrographic analyses for any unusual trace elements that might be present in the palmar skin surface film of "rusters" were carried out in this study in the following manner.

Subjects washed their hands for two minutes with pHisoHex,† rinsed thoroughly with water and then rinsed three times with copious amounts of distilled water. Hands were then patted dry with paper towels. Samples of sweat were collected approximately 30 minutes later by dusting spectrographically pure lithium carbonate-graphite matrix over the palmar surface of the hand. The matrix was rubbed gently over the surface of the skin, collected and placed in a graphite electrode, and burned in the emission spectrograph. The spectra were recorded on Kodak Spectrum Analysis No. 1 Plates.

†pHisoHex—An antibacterial, degerming, emollient, sudsing detergent. Available through Winthrop Laboratories, New York 18, N. Y.

Micrograms of Element in the Sample

Sample	Date	Sodium	Magnesium	Iron	Silicon	Aluminum	Copper
Control	4/12/57	2	0.08	0.01	0.2	0.3	0.008
Ruster	4/12/57	3	0.1	0.08	0.8	0.4	0.01
Control	4/23/57	3	0.1	0.05	0.2	0.2	0.008
Ruster	4/23/57	4	0.05	0.1	0.4	0.4	0.08
Control	4/30/57	2	0.1	0.03	0.6	0.2	0.005
Ruster	4/30/57	3	0.05	0.3	0.1	0.3	0.008
Control	5/16/57	2	0.1	0.1	0.3	0.2	0.003
Ruster	5/16/57	3	0.08	0.08	0.2	0.2	0.005
Control	5/23/57	1	0.1	0.1	0.4	0.5	0.01
Ruster	5/23/57	2	0.08	0.1	0.3	0.8	0.08

The values determined are semiquantitative and are based on visual estimations made from previously prepared standards. The control was a subject from Group II to be classified later; the "ruster" was classified in Group IV.

The following metals were tested for but not detected: lead, potassium, cobalt, arsenic, phosphorus, bismuth, boron, tin, nickel, manganese, calcium, antimony, barium, and chromium. As far as could be determined, there were no unusual trace elements found. The sodium concentrations found in the sweat of the "ruster" were higher throughout. Thus the relationship of higher sodium and subsequently higher

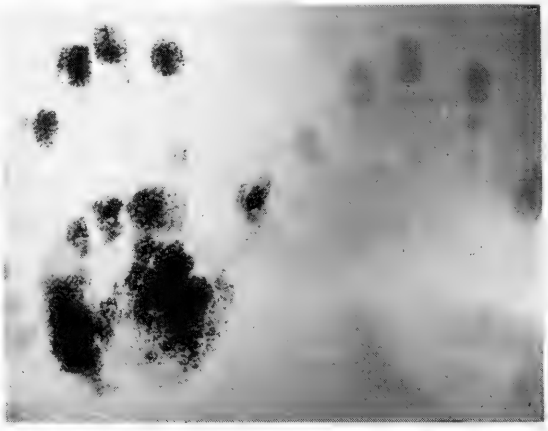

Fig. 2—Polished mild steel surface showing hand print of a "ruster" on left and subject selected from Group I on the right. Photograph taken three months after plate was touched. Both subjects washed hands for two minutes with pHisoHex, thoroughly rinsed in water, and then rinsed three times with plenty of distilled water. Twenty minutes was allowed to elapse before the prints were made. Hand contact was sustained for approximately two minutes' time with the surface of the plate. Hand print on right shows no corrosion of metal—skin surface film was readily removed from the plate using acetone as a solvent.

chloride to rusting is the important finding.

In addition, Collins has shown that when urea and lactic acid (normally present in sweat) are tested for their ability to produce corrosion on mild steel, each is found to produce less than 5% of the amount of corrosion caused by the same concentration of sodium chloride solution. Collins has also demonstrated that the addition of lactic acid to test concentrations of sodium chloride produces somewhat more corrosion than is produced when the same concentration of sodium chloride is used by itself. Contrawise, he has shown that when an emulsion of oleic acid in water was added to the test concentration of sodium chloride, less corrosion was produced.

The Transfer of Skin Surface Film and Its Effects on Steel Plates

Fig. 2 shows the surface of a polished, degreased steel plate; firm contact between the plate and the subjects' hands was maintained for two minutes. The palm and finger prints on the left were made by a "ruster." It shows severe corrosion maximally distributed in areas where palmar sweating is noted regularly and profusely. Corrosion can often be noted beginning as a series of pinpoint specks of brownish material on the metal where the palmar aspect of the tips of the fingers rested. The same pattern can be demonstrated as being the sites of the excretory pores of eccrine sweat glands using the starch-iodine technique frequently described in dermatologic literature. During the next several days, the brownish specks became confluent. Where sweating is especially profuse and the hand is uniformly wet, the sweat pore nidus pattern of chloride corrosion is often missed, and corrosion begins on the metal surface in patches of brownish-staining material. X-ray diffraction analysis of the corrosion material itself, three months after palmar sweat had been deposited on the metal plate, revealed it to consist essentially of ferric oxide and one molecule of water.

The palm and finger prints on the right were taken from a person who rarely has shown any evidence of palmar sweating. It consisted largely of skin lipid surface film and could be removed readily with lipid solvents. There was no evidence of corrosion on the metal surface when the lipid film was removed. Both of these prints were taken 20 minutes after the hands had been washed with pHisoHex skin cleaner for two minutes followed by three profuse rinses with distilled water. Metal plates were exposed, without covering, in a room where the temperature was maintained at 76°F and the relative humidity was approximately

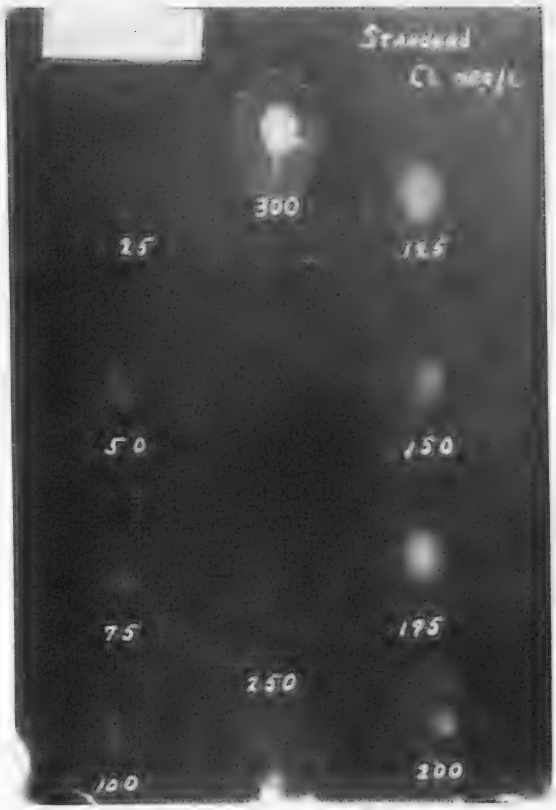

Fig. 3—The tip of the clean, dry finger was dipped into sodium chloride solutions of the strength as indicated, and then touched to the surface of the impregnated gelatin plate. Photographs were taken with Royal Pan Film using a Wratten #61 Filter. This plate was subsequently used as a master plate.

47%. (The rate of corrosion can be speeded up initially by increasing the temperature and humidity, particularly the latter.)

In both of the above patients, the pH of the palms and palmar surface of the fingers measured 4.5 to 5.0 using a glass electrode. Surface pH was measured in several "rusters" but was never found lower than 4.5. This latter value was frequently found in persons whose skin surface film caused no corrosion of the metal surface. Contrawise, Collins has demonstrated that artificial sweat solutions produce somewhat more corrosion at pH 5.2 than at pH 8.0. It is felt, however, that from a practical point of view sweat pH plays a very minor role in corrosion of metal. This finding is of importance when it is realized that most plant personnel talk about the acidity of sweat as being the cause of corrosion of metal.

It is reasonable, therefore, to conclude that chloride ion released in eccrine sweat and transferred to the metal surface in a fluid vehicle, such as occurs when the metal is being

Figs. 4 and 5—Hand print on impregnated gelatin plate (left) and on metal plate (above). Subject selected from Group II. Shows just-detectable corrosion. Chloride range 50-100 mEq per liter when compared with "master plate."

handled, leads to the major portion of subsequent corrosion of the metal. Futhermore, it is well to point out that palmar sweating occurs following nervous or emotional stimuli and is not ordinarily provoked by thermogenic stimuli.

The Classification of a Population by Metal Plate Corrosion Tests

In testing the ability of individuals to produce corrosion of mild steel plates, it was observed that it was possible to divide them on the basis of their reactions into four groups which correlated to some degree with their palmar sweat patterns. The first group was made up of subjects whose palms were always dry. This group transferred a print of skin surface film to the metal plate which, for practical purposes, caused no corrosion. The second group of subjects produced just-detectable corrosion on the metal surface. This group stated that they noticed moist palms on occasion. Test results on these people were not constant, in that at times they produced just-detectable corrosion and at other times no corrosion. The third group of subjects stated that their palms were frequently moist, particularly after psychic stimuli. This group produced detectable to severe corrosion when tested at various times. The fourth group of subjects — the "rusters" — produced severe corrosion on every test. They stated that their palms were rather moist practically all the time. In addition, some of these subjects were aware of "bursts of sweating" occurring after psychic stimuli. Four of this latter group knew definitely that they produced corrosion of metal just by touching its surface.

Case Histories of Typical "Rusters"

The first subject was a machinist apprentice who had received two years of general training before starting to work on inspection. Within a few weeks' time after he began handling finished work he was found to be a "ruster." This subject was restricted thenceforth from inspecting and handling dies or vernier gauges.

The second subject was also aware of his propensity to corrode metal. He was able to get along fairly well as a general machinist for several years by keeping a thick film of lubricating oil on his hands while he worked. He had an opportunity to accept a better position as a locksmith but found that, despite extreme care, the tumblers in the lock mechanism which he handled were corroding and the locks did not function properly.

The third subject knew of his rusting capacity. For some years he had been buying his own micrometers because these corroded so rapidly. He pointed out that supervision ob-

Figs. 6 and 7—Hand print on impregnated gelatin plate (left) and on metal plate (above). Subject selected from Group III. Shows detectable corrosion. Chloride range 100-150 mEq per liter when compared with "master plate."

served the operators' micrometers to detect those who might cause corrosion of metal. His regular work consisted of machining brass and aluminum, and he was able to get along quite well at this type work. However, a decrease in work necessitated that he machine steel. Within a few months on this new work, he was identified as a "ruster" by supervision and certain work restrictions were applied.

The fourth subject, who had been restricted from working with mild steel for some years, had an opportunity to work as a setup operator making printed circuits. There was considerable question on the part of supervision as to whether he would be able to continue in this work because severe corrosion was noted on the thin copper plating used in the makeup of the circuits.

All of the subjects mentioned above showed no abnormalities on physical examination. Medical history was negative. They were all in good health. There were no developmental or nutritional defects noted during early childhood. Their diet was average or normal. Salt intake was not excessive. Laboratory work and chest x-ray examination[3] showed no abnormalities. Three of the subjects were married and had normal children; the fourth man was unmarried. Repeated questioning on complaints referable to the digestive or respiratory tract revealed no abnormalities. We were unable to detect any history of cystic fibrosis of the pancreas in them or in their immediate families.

A Simplified Screening Test

It seemed possible to detect the "ruster" in industry with the use of polished metal plates. However, this requires several weeks for the completion of the test. In addition, the preparation, polishing, handling, and storage of the plates is time-consuming and expensive. To shorten the time required in detecting the "ruster," electrical conductivity devices were investigated to measure the electrolyte concentration on skin surface. Results utilizing this technique necessitated considerable interpretation.

An increase in the quantity of chloride ion present in palmar sweat has been demonstrated as being the primary abnormality in "rusters." This same abnormality has been described in patients with pancreatic cystic fibrosis. This disease is inherited as a recessive trait. It involves the exocrine glands, especially those secreting mucus. Most characteristically, the pancreas, respiratory system, and sweat glands show disturbed function beginning in infancy and typified by malnutrition, pancreatic insufficiency, and respiratory infection. Sweat from

Figs. 8 and 9—Hand print on impregnated gelatin plate (left) and on metal plate (above). Subject selected from Group IV. Shows severe corrosion. Chloride range 150 mEq per liter plus when compared with "master plate."

cystic fibrosis patients may contain upwards of 150 mEq of chloride per liter. Shwachman[4] and others[5, 6] have described simple tests for the detection of increased chloride in sweat to aid in the diagnosis of cystic fibrosis.

It seemed probable that such a procedure could be utilized for the early detection of "rusters." The connection between the two conditions—cystic fibrosis and "rusting"—may not be as improbable as would seem on initial consideration. Shwachman has found approximately 20% of the parents of children with cystic fibrosis of the pancreas to have increased concentrations of sodium and chloride in their sweat, without other manifestations of the disease.

Glass plates measuring 5 by 7 inches were coated with a thin layer of essentially salt-free gelatin impregnated with silver nitrate and potassium chromate. Plates were separated from each other by a thin sheet of plastic, placed in a plastic container, and refrigerated at 34°F. When removed for test, the gelatin plates presented a moist, soft surface. Contact of chloride ion with the impregnated gelatin surface produced a whitish (silver chloride) to yellowish color (sodium chromate) against the red background of the plate. This reaction may be expressed chemically as follows:

$2AgNO_3 + K_2CrO_4$—Ag_2CrO_4 (ppt) $+ 2KNO_3$—impregnated gelatin plate

$Ag_2CrO_4 + 2NaCl$—$2AgCl$ (ppt) $+ Na_2CrO_4$—chloride test

A master plate (Fig. 3) was prepared using known concentrations of chloride ion to facilitate interpretation of the test. Subjects were again placed in the four groups previously mentioned, but now on basis of the color change noted, as follows:

Group I—Less than 50 mEq/liter of chloride ion. (No corrosion.)

Group II—50-100 mEq/liter of chloride ion. (Slight detectable corrosion.)

Group III—100-150 mEq/liter of chloride ion. (Detectable corrosion.)

Group IV—150 mEq/liter and above of chloride ion. (Severe corrosion.)

Preparation of Subject to be Tested

1. Inspect palmar surface of hands and fingers to rule out any obvious skin disorder. Subjects with obvious skin trouble were not tested.
2. Wash hands for two minutes time with pHisoHex and thoroughly rinse.
3. Flush hands thoroughly three times with distilled water.
4. Pat palmar surface of hands dry using a fresh, clean, cotton surgical towel.

5. Seat subject comfortably and instruct him (or her) not to touch anything with either hand. Hands are preferably held upward with palms directed forward.

6. At the end of 20 minutes, press subject's hand gently and firmly on the gelatin plate. Either hand may be used for testing.

7. Read results of the test directly on comparison with master plate, and place subject in appropriate group.

The test is relatively simple to perform and little training is required by the observer to classify the subject properly. Figs. 4-12 show the results in several subjects tested. Correlation with corrosion on metal plate surfaces is shown in certain of the subjects.

It is of considerable interest to note that the "rusters" appear to be in a class by themselves as far as palmar sweat chloride concentration is concerned. Moreover, no evidence of cystic fibrosis, adrenal insufficiency, panhypopituitarism, or nephrogenic diabetes insipidus —all of which cause an elevated chloride in sweat — could be found.

Fig. 10—Hand print on metal plate. Subject (child) with fibrocystic disease of pancreas on left—Impregnated gelatin plate test showed chloride range 100-150 mEq per liter. Subject placed in Group III. Hand print on right from parent (mother) of subject. Impregnated gelatin plate test showed chloride range 50-100 mEq per liter. Subject placed in Group II.

Fig. 11—Hand print on metal plate. Subject (child) with fibrocystic disease of pancreas on lower left. Impregnated gelatin plate showed chloride range 50-100 mEq per liter. Subject placed in Group II. Hand print on metal plate—upper portion with thumb not showing of parent (father) of subject. Impregnated gelatin plate showed chloride range 0-50 mEq per liter. Subject placed in Group I.

The Prevention of Rusting

Preventive techniques to reduce corrosion of mild steel may be outlined as follows:

1. At the time of preplacement examination, all personnel required to work in critical areas should be screened. The "ruster" is readily detected.

2. Employees already working in critical areas where corrosion is a problem should be screened and supervision advised of the results. In special critical areas, day-to-day screening may be required.

Prevention of Rusting in "Normal" Persons

The following techniques can be used to reduce corrosion resulting from subjects in Groups II and III.

1. Avoid *excessive* salt intake. It has been shown by Locke[7] that at a constant rate of sweating the chloride concentration parallels the salt intake. This would certainly have some effect on those with profuse palmar sweat. It would help only slightly on those with a slow intermittent sweat pattern, as studied by Lobitz.[8] However, most subjects in this latter class would initially be classified as being members of Group IV.

Fig. 12—Micrometer used by a "ruster" from Group IV. Photograph taken after one week use to show corrosion. Small rectangular piece of metal was held by "ruster" for 30 minutes' time. Corrosion noted in 24 hours. Photography taken about one week later.

2. Wash hands frequently. Both the *rate* and the *duration* of sweating influence the concentration of chloride on skin. Frequent washing is a practical expedient in the removal of salt.

3. Provide a cool working environment with controlled humidity. Special critical areas may well require air conditioning. Temperatures of 65-70°F and relative humidity of 50% are suggested. It has been shown that the higher the skin surface temperature, the higher is the chloride content[9] of its surface film.

4. Provide an ample supply of drinking water. In a controlled working environment (as mentioned above in 3) where no unusual salt loss may be expected through profuse sweating, ample drinking water has been shown[10] to depress the concentration of chloride in sweat. Without control of the working environment, this method would not be feasible.

5. Frequent applications of one of the various types of hydrophilic ointments presently available on the market to the hands appears to have possibilities for short-term protection and further investigation is advisable. This technique, of course, would be limited to those industries where application of ointments to the employee's hands does not create a greater secondary problem.

6. Items of protective clothing such as cotton gloves that may be changed frequently, rubber gloves that need be worn for only short periods of time, and other mechanical devices may all aid in reducing corrosion. These special situations require individual evaluation.

7. The administration of atropine-like drugs by mouth is not practical. To reduce sweating appreciably, the side-effects experienced by the subject, such as dilation of the pupil and dry mouth, are so uncomfortable that the use of drugs now available is not advised.

8. The use of antiperspirants not containing chloride ion applied locally to the hands, under medical supervision, has possibilities. However, constant watchfulness by plant supervision would be required, since there could be an increase in skin disorders of the hands due to sweat retention. Usually, such practice is not feasible for long-term situations.

9. Theoretically, more corrosion of metal should be expected during the winter months because sweat contains about twice as much salt during this period as it does through the summer.[11, 12] This finding probably applies in part to palmar sweating and is usually explained as a special case of acclimatization. In actual plant practice, however, an apparent paradox occurs. Most supervisors stress the fact that corrosion of metal appears to occur more frequently and more severely during the summer. There are, at least, two factors which support their contention. The first is that machine shops are ordinarily not air-conditioned. Subjects sweat a great deal in this environment, and there is considerable mechanical transfer of salt from other exposed parts of the body by the hands to metal surfaces. The second factor is that the rate of corrosion on the metal surface is speeded up by the higher temperature and higher relative humidity present during the summer months.

10. Both sexes have the problem of causing corrosion of metal. The younger age groups, with seemingly more labile emotions, present the problem more frequently. Cyclic variations related to menses as regards eccrine sweat production, chloride concentration, and emotional lability have not been studied sufficiently to justify the drawing of any general conclusions. It would seem that these variations would require considerable individualization with day-to-day or frequent spot checking with the impregnated gelatin plates.

Summary

This article considers the problem of the "ruster" in industry. The ramifications of this problem may have application in any industry where perspiration can be shown to have a deleterious effect on finished product. The component, chloride ion, present in eccrine sweat and causing the major portion of corrosion on mild steel is determined. Transfer of this component is considered. A rapid test for screening subjects to detect the "ruster" is outlined. No relationship between the "rusters" studied in this series and systemic disease such as pancreatic fibrosis could be determined. Certain preventive techniques are considered.

Acknowledgments

In the preparation of this paper, we are indebted to Dr. J. Radebaugh, Pediatrician, of Rochester, N. Y., for making available his patients with pancreatic fibrosis; to Mr. G. Boomer for technical assistance in the preparation of the impregnated gelatin plates; to Mr. A. Wittmer for photographing the gelatin and metal plates; and to S. Foster, C. Zuehlke, and R. Griffith, all of Eastman Kodak Company, for certain chemical analyses.

(Dr. Buckley's address is Eastman Kodak Company, Rochester, N. Y.)

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The Tolerance of the Skin to Lubricating and Cutting Oils

Geoffrey Hodgson, M.B.E., M.D., Cardiff, Wales

Nature of the Materials

The lower-boiling distillates of crude petroleum are usually regarded as defatting agents and skin irritants. The lubricating oil fraction is considered as being relatively well tolerated by the skin.

Lubricating Oils and their Additives

Lubricating oils are made from the stabler hydrocarbons, either crude oil residues (heavy lubricating oils) or the ordinary distillation cut (light lubricating oils). From these derive the liquid oils for engine lubricants, the cutting oils, and the solid greases, where the oil is thickened by incorporation of a lime or soda soap. Engine lubricating oils consist of spindle oil (pale amber, thin), engine oils (red, medium consistency), and cylinder oils (green/black, thick). Differing proportions of these yield the various grades.

Under heat and pressure lubricating oils oxidize, forming acid peroxides. The oils become semisolid or solid with engine carbon. Special additives may be incorporated to inhibit oxidation or to achieve a special effect, e.g., antioxidants, detergents, and antirust. Detergents are really dispersants which keep engine carbon in suspension, so that it will not settle on engine parts and, being slightly basic, also combine with the oxyacids formed by oxidation. They are used in diesel and some gasoline engine lubricants. Rust preventives emulsify the moisture to prevent contact with the metal, and some surface-active compounds are adsorbed on the metal as a barrier to rust. They are used for special types of machinery as turbocompressors and exhausters, hydraulic machinery, and in gear and bearing oils. Examples of other additives are those for foam suppression and nondrip fatty oils for textile machinery. Many of these additives are secret formulae but may have the following general makeup:

Antioxidants—(1) High-temperature uses (e.g., internal combustion engines), sulfur or phosphorus compounds or combinations as sulfurized esters, terpenes, aromatic and alkyl sulfides, aryl or alkyl phosphites, and metallic salts of dithiophosphoric acid; and (2) low-temperature uses (e.g., turbine oils), amines, and hydroxy or phenolic compounds, such as 0.1% phenyl-α-naphthylamine.

Detergents—Commonly calcium, barium, magnesium, aluminum, or tin in combination with a sulfonate (SO_3H), hydroxyl (OH), carboxyl (COOH), or mercaptan (SH) group, which are combined with a high-molecular-weight straight or branched paraffin chain or an aromatic or naphthenic ring.

Antirusts—Neutralized sodium or calcium soaps from petroleum sulfonates or wool grease (to which pine oil may be added as a deodorant) for moisture emulsification; or special antirust organic reducing agents of amine or hydroxy types, usually under patent.

Cutting Oils

These may be simple fluids like water or soap, kerosene, and mineral and lard oils, but are mostly of two types: soluble (emulsion suds) oil, and insoluble cutting oils.

Soluble oils are mixtures of spindle oil and soaps (petroleum sulfonates) to which are added disinfectants, or resin soaps; oxidation inhibitors (hydroxy compounds or amines); proprietary antiseptics; and water hardeners or softeners, as sodium hydroxide or carbonate, trisodium phosphate, or borax.

Four common types of insoluble oils are

(a) Straight mineral oils: spindle and colza oils and kerosene.
(b) Fatty oils: lard and rape oils.
(c) Mixtures of mineral, animal, or vegetable fatty oils.
(d) Extreme pressure oils (E.P.)

The E.P. oils may comprise mineral or lard oils sulfurized with free (active) or combined

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(inactive) sulfur, or they may be chlorinated or sulfochlorinated. Petroleum oils naturally contain some combined sulfur, and Middle East petroleum has more sulfur than Pennsylvanian. Sulfur imparts an "affinity" for metal and lowers surface tension. Sulfurized and chlorinated oils have antiwelding properties and lower the temperature of cutting operations.

Cutaneous Effects of Oils

Klauder and Brill[1] by single patch tests using kerosene, mineral seal oil, and light spindle oil showed a correlation between the boiling range and the irritant action which diminished as the intermediate distillate with a "twilight of irritability" approached the well-tolerated lubricating oil. All reactions were considered primary irritant. Lubricating oils are seldom recorded as irritant. Twiston Davies[2] reported positive patch tests in only 2 of 20 patients who alleged that their eruption was due to oil "allergy," but no information was given of the composition of the oil. The good reputation of tolerance for the lubricating oil fraction may, however, have become more suspect by additive compounding. Dermatitis has been reported from an antioxidant used in a motor lubricating oil.[3]

Cutting oils have a darker reputation and are a frequent cause of dermatitis,[4] constituting the origin of 15% of all occupational dermatoses.[5] Soluble oils cause soap-like softening and eczematization, but dermatitis may result from the solvent effect of the oil fraction, high pH,[6] rancidity (e.g., lard oils), added chemicals (e.g., the phenolic disinfectants), and perfumes; while oxidation inhibitors are potential sensitizers. Insoluble oils are said to be more offensive than emulsion oils.[7] They block hair follicles and cause oil acne (folliculitis). Squire[8] reported an 80% incidence in an engineering factory. Pyogenic infection may lead to spreading infective dermatitis. Sulfurized and chlorinated oils are said to cause more skin trouble than most lard oils,[9] and chloracne can occur. Contamination by swarf in all cutting oils can cause mechanical injury with secondary infection. This varies from the minute punctures of fine aluminum or cast-iron dust difficult to remove, to the penetrations of sharp needles of brass and to razor-like incisions of spiral steel strips. Adherence of swarf to natural skin folds and creases or wipe rags by insoluble oil is a particular hazard. Pyogenic infection of cutting oils is not considered to be a primary cause of dermatitis.[10] Soluble cutting oils become more contaminated with use, insoluble oils remaining pretty free. Overheating of cutting oil in the presence of metals results in chemical reactions and the formation of irritants such as acroleates,[9] and may lead to overheating of the skin,[11] which circumstances may cause harmful effects beyond those aforementioned.

Methods of Study and Observations

The tolerance of normal and eczematous skins to samples of lubricating, E.P. (insoluble), and soluble cutting oils of known composition, and of crude tanker sludge was determined by patch test exposure. An attempt was made to estimate the possible effect of usage on the skin reaction and particularly the role of oxidation and metallic contamination, and the sensitizing capacity of additives. Some six drops of oil were applied by prepared patch test to the flexor aspect of the upper arm and forearm. Three types of test were employed:

1. *Prophetic Patch Test*[12]—A 24-hour test with challenge 14 days later and follow-up for delayed reactions.

2. *Repeated Insult ("Fatiguing Reaction") Test*[13]—Repeated insult applications on the same area (marked by a small dot of brilliant green to ensure accuracy) for 24 hours on and 24 hours off for 1 month, 15 or so being the average number of insults. Testing of an area ceased when a positive reaction occurred. A 48-hour challenge was performed on the same area after two to three weeks' rest.

3. *Forty-Eight Hour Test*—Patch tests with the individual additives in liquid petrolatum, as well as liquid petrolatum as a control, were performed in all persons with positive reactions and in most with negative responses to the oils.

Results were recorded as + (redness), ++ (redness and swelling papules), +++ (intense redness, swelling, papules, vesicles), ++++ (confluent blisters), and +++++ (desquamation, oozing, necrosis). No positive reactions were obtained in any series over ++ in this system of scoring. Completed challenge tests and the optimum 15 insults were not always possible.

Two groups of subjects were tested: persons with normal skins, and persons with eczematous skins. Some took part in more than one experiment. The normal group, with twice as many men as women, included medical staff personnel, students, nurses, technicians, and some patients with acne and psoriasis. The eczematous group all had active eczematous dermatitis of various origins and varying degree somewhere on the body. The arms were clear for testing, although the hands might be affected. Testing was done on intact skin.

Adhesive plaster reactions of two types were noted: In one type contact dermatitis necessitated a change of the plaster or, in cases with vigorous reactions, the test was stopped. In the second type of reaction noted during insult testing, the skin appeared to harden after 7 to 10 days.

Lubricating Oils: Steam Turbine and Electrical Transformer Oils

Experiment I—The turbine oil was a straight amber-colored, naphthenic spindle oil of viscosity range in Redwood seconds at 140°F 70″ to 140″ (as compared to high-temperature oxidation of internal combustion engines); the transformer oil being highly refined, almost white spindle oil, Redwood viscosity at 70°F of 120″. The antioxidant additive was 0.1% phenyl-α-naphthylamine. These oils were tested:

1. Straight steam turbine oil
2. Straight turbine oil + antioxidant
3. Straight turbine oil + antioxidant, after use (oxidation)
4. Straight transformer oil
5. Straight transformer oil + antioxidant
6. Straight transformer oil + antioxidant, after use (oxidation).

To simulate the effects of use, oxidation was carried out experimentally by blowing air through the oil at a rate of 15 liters per hour at 110°C for 300 hours and then for 300 hours at 120°C using copper and steel foil as catalysts. Acidity levels were from 0.28 mg to 0.56 mg KOH per gram. The samples were quite clear and free of metallic contamination. The distribution of reactions using the "prophetic" as well as the repeated insult test is shown in Tables I, II, and III.

Of the three subjects demonstrating positive reactions one gave positive results to all except straight turbine oil, e.g., oils 2, 3, 4, 5, and 6 after two insults; one to oils 4 and 5 after four insults; and the other to oils 3 and 6 after seven insults. The average number of insults which provided reactions was 4.3. More reactions (9:1) occurred on the repeated insult than on the 48-hour challenge, only one positive being reproduced on 48-hour challenge (Table II). No positive reaction occurred in either group with the additive. Only one more positive reaction occurred with oxidized turbine oil than with unoxidized, and the same number occurred with the transformer oils. It appears that turbine oils and electrical transformer oil before and after use produced no irritant effect on normal skin but caused reactions in 3 out of 10 (30%) of eczematous skins. There was no evidence of sensitization to the antioxidant 0.1% phenyl-α-naphthylamine. The irritant effect was not significantly altered by oxidation. Repeated insults produced more positive results than the 48-hour challenge.

Insoluble Cutting Oils

Experiment II—Three samples of sulfurized and chlorinated insoluble cutting oils (E.P.) containing a 0.1% copper-staining inhibitor additive, both before and after machine use, were tested. The samples are listed below:

A. Mineral oil + 0.5% sulfur + copper-staining inhibitor

Table I—Reaction of Normal Subjects Exposed by Prophetic Patch Test to Lubricating Oils

Test	Subjects No.	Results Positive %	Results Negative %
Primary exposure	63	0	100
Second exposure (24-hour challenge)	44	0	100

Table II—Reactions of Normal and of Eczematous Subjects Exposed by Repeated Insult Test to Lubricating Oils

			Reactions: Lubricating Oils (Various Preparations)				Additives Only	
			Repeated Insult		48-Hour Challenge		Repeated Insult and Challenge	
Group	Subjects No.	Insults Av. No.	Positive %	Negative %	Positive %	Negative %	Positive %	Negative %
Normal	14	14	0 (14)	100 (14)	0 (14)	100 (14)	0 (14)	100 (14)
Eczematous	10	10	30* (3)	70 (7)	10 (1)	90 (9)	0	100 (10)

Numbers in parentheses indicate numbers of subjects.
*Positive reactions occurred after an average of 4.3 insults.

*Table III—Total Number of Positive Reactions to Lubricating Oils**

	No.	48-Hour Challenge	Additive
1. Straight turbine oil	0	0	0
2. Straight turbine oil plus additive	1	0	0
3. Straight turbine oil plus additive oxidized	2	0	0
4. Straight transformer oil	2	0	0
5. Straight transformer oil plus additive	2	1	0
6. Straight transformer oil plus additive oxidized	2	0	0

*These reactions occurred in three subjects with eczematous skin exposed by the repeated insult technique.

B. A after use
C. 93% mineral oil + sulfurized sperm oil + chlorinated petroleum fraction
D. C after use
E. 73% mineral oil + sperm oil + chlorinated petroleum fraction + sulfurized sperm oil
F. E after use.

One used sample (D) appeared to be much more heavily contaminated with metallic dust than the other used oils (B and F).

The results of prophetic and repeated insult tests in normal and eczematous subjects are shown in Tables IV, V, and VI. In the prophetic test the same person gave positive reactions to oils D and F on initial and challenge tests. Excluding one eczematous person who reacted to all oils on the first exposure, the average number of insults required to produce reactions was: A, 6; B, 5; C, 8; D, 4; E, 4.75, and F, 5.3. For all the E.P. materials the average was 5.5 insults.

Irritant effects occurred in both normal and eczematous skins, a third of the latter showing lowered tolerance. All the samples of oil produced irritant effects in eczematous persons but only two oils, sample D (93% mineral oil + sulfurized sperm oil and chlorinated petroleum fraction) and sample F (sulfochlorinated oil), produced a reaction in the normal group. Both were contaminated used oils. Most reactions were also produced in eczematous skins by oil D. The degree of contamination of this sample had been noticed before testing. The irritant effect, comparing before and after use, was unchanged by use in all oils except this particular used oil (D). This oil also produced the earliest reaction on repeated insult—average 4 insults. The more highly sulfurized and chlorinated oil produced the next highest number of reactions. Twice as many positive reactions occurred in eczematous skins with the more highly chlorinated and sulfurized oil (73% mineral oil + sperm oil + chlorinated petroleum fraction + sulfurized sperm oil).

From these observations it appears that positive reactions were produced in normal skin by used and contaminated samples of sulfochlorinated oils, but not by the fresh oils or by mineral oil with 0.5% sulfur. A greater number of positive reactions occurred in eczematous skins than in normal skins. Eczematous skins reacted both to sulfurized and to sulfochlorinated oils. There was no evidence of sensitization to the copper-staining inhibitor additive in normal or eczematous skins. Most reactions in all skins occurred with the most highly contaminated used oil. Excluding the contaminating effect, the most highly sulfochlorinated oil produced the more irritant reactions. More positive reactions occurred on repeated insults than on the 48-hour challenge test. The average time for all oils to cause irritation was 5.5 alternate 24-hour insults, the most highly contaminated oil producing the earliest irritant effects, followed by the more highly sulfochlorinated oil.

Soluble Cutting Oils

Experiment III—The mineral oils in this experiment were naphthenic spindle type, amber or light red in color, with Redwood viscosity about 200″ at 70°F. Three samples were tested, as shown below:

G. Emulsion oil with 4.4% cresylic acid, 1:20 water dilution

Table IV—Reactions of Normal Subjects Exposed by Prophetic Patch Test to E.P. Oils

Test	Subjects No.	Results Positive %	Results Negative %
Primary exposure	63	0	100 (63)
Second exposure (24-hour challenge)	45	0	100 (45)

Numbers in parentheses indicate numbers of subjects.

Table V—Reactions of Normal and of Eczematous Subjects Exposed by Repeated Insult Test to E.P. Oils

Group	Subjects No.	Insults Av. No.	Reactions: E. P. Oils: Repeated Insult Positive %	Repeated Insult Negative %	48-Hour Challenge Positive %	48-Hour Challenge Negative %	Additives Only: Repeated Insult and Challenge Positive %	Additives Only: Repeated Insult and Challenge Negative %
Normal	11	11	9 (1)	91 (10)	9 (1)	91 (10)	0	100 (11)
Eczematous	26	12	35 (9)	65 (17)	23 (6)	77 (20)	0	100 (26)

Numbers in parentheses indicate numbers of subjects.

Table VI—Total Number of Positive Reactions to E.P. Oils Among Normal and Among Eczematous Subjects

Oils	Positive Reactions No.	Positive 48-Hour Challenge No.	Negative 48-Hour Challenge No.	Positive Reactions to Additives No.
A	2	1	1	0
B	2	2	0	0
C	2	2	0	0
D	10	7	3	0
E	7	4	0	0
F	4	4	0	0

H. Emulsion oil with 3% xylenols, 1:20 water dilution
I. Emulsion oil with 8% xylenols, 1:40 water dilution.

The blends contained special soaps for partial water solubility, the hydroxy compounds (cresylic acid or xylenols) assisting in the oil/soap stability. The cresylic acid included isomeric cresols (ortho-, meta-, and para-); the xylenols, isomeric xylenols and higher homologues of the same series. In this series the antioxidant additive, 0.1% phenyl-α-naphthylamine, was also tested as a control. The results are shown in Tables VII and VIII.

On the average 6.1 insults were required before an irritant effect occurred (G, 4.25; H, 7.25, and I, 7.0 insults). Positive reactions occurred in both normal and eczematous skins, eczematous skin again showing considerably less tolerance than normal skin: 36% positive reaction (six persons) as compared to 4% (one person). More positive reactions occurred on the repeated insult than on the 48-hour challenge, 14:1. One positive reaction occurred later on 48-hour challenge, in a normal skin, that had not previously occurred on the repeated insult test. The xylenol emulsion oil produced the most reactions in the eczematous group; but the cresylic acid emulsion oil produced an earlier irritant effect, requiring four insults as compared with seven of the other oils. No positive reactions occurred to 0.1% phenyl-α-naphthylamine antioxidant.

Table VII—Reaction of Normal and of Eczematous Subjects Exposed by Repeated Insult Test to Soluble Cutting Oils

			Reactions					
			Soluble Cutting Oils				Additives	
			Repeated Insult		48-Hour Challenge		Repeated Insult and Challenge	
Group	Subjects No.	Insults Av. No.	Positive %	Negative %	Positive %	Negative %	Positive %	Negative %
Normal	25	8	4 (1)	96 (24)	4 (1)	96 (24)	0	100 (25)
Eczematous	13	12	46 (6)	54 (7)	8 (1)	92 (12)	0	100 (13)

Numbers in parentheses indicate numbers of subjects.

Table VIII—Total Number of Positive Reactions to Soluble Oils Among Normal and Among Eczematous Subjects

		Repeated Insult		
Oil	Blend	Normal	Eczematous	48-Hour Challenge
G	4.4% cresylic acid oil, 1:20 water	1	3	0
H	3% xylenol oil, 1:20 water	1	3	1
I	8% xylenol oil, 1:40 water	0	6	0

Table IX—Reactions of Normal and of Eczematous Subjects Exposed by Repeated Insult to Crude Oil Residue

			Reaction to Crude Oil Residue			
			Repeated Insult		48-Hour Challenge	
Group	Subjects No.	Insults Av. No.	Positive %	Negative %	Positive %	Negative %
Normal	22	8	0	100 (22)	0	100 (22)
Eczematous	10	11	0	100 (10)	0	100 (10)

Numbers in parentheses indicate numbers of subjects.

The observations made with soluble cutting oils indicate that most reactions occurred in eczematous subjects. Six of the eczematous reacted (36%) in contrast to one of the normal group (4%). More positive reactions occurred on the repeated insult test than on 48-hour challenge. Doubtful positive reactions followed by definite responses to insult retesting after subsidence of positive reactions indicated varying day to day tolerance of the skin or possible attempts to harden against the irritant.

Crude Oil Residue

Experiment IV—The crude oil residue from cargo tanks of tankers bringing crude Middle East oil was also tested. This was a thick mud-like sludge scraped from the cargo tanks after the oil had been pumped out. The results of exposure by repeated insult tests, shown in Table IX, were entirely negative.

Discussion

The results are those which might be expected from previous work and clinical impression. Subjects with normal skin show a complete tolerance to new and used (oxidized) refined spindle oil for turbine and electrical transformer lubrication even after long-continued exposure. Cutting oils had these effects after continued exposure: soluble oils produced a few irritant reactions; but none occurred with clean, compounded insoluble cutting oils of E.P. type, either sulfurized or sulfochlorinated. The only irritant effects were recorded with used sulfochlorinated oils which had been contaminated by metal.

The intact skin of persons with active eczem-

atous lesions elsewhere, on the contrary, showed a marked lack of tolerance to the lubricating and cutting oils tested. Continued exposure produced a remarkably constant percentage of positive reactions to all the types of oils, e.g., turbine and electrical transformer oils, 30%; E.P., insoluble cutting oils, 35%; and soluble "suds" cutting oils, 36%. In the compounded cutting oils the most highly sulfochlorinated E.P. oils seemed the more irritant, but irritant reactions occurred also to simple sulfurized oils. The lack of tolerance to oil itself even extended to the simple refined spindle oil of the turbine and transformer oils. In lubricating oils it would seem that simple oxidation by use alone (using oxidized oils) did not influence at all the irritant effect, but that other factors, such as the degree of metallic (steel) contamination, may have increased the number of irritant reactions of E.P. cutting oils in eczematous skins.

The results could be taken to indicate that a completely intact epidermis has a very good tolerance to refined spindle oils for lubrication (turbine and transformer oils) and to clean compounded sulfurized or sulfochlorinated E.P. cutting oils. This tolerance may be lowered in eczematous subjects in whom skin damage has occurred previously by trauma from contaminating metal particles or by primary epidermal damage from the continued effect of the aqueous soap emulsions in the soluble cutting oils. Once the lowered tolerance is established by eczematous damage, then irritant effects can occur even with the refined spindle oils as used for turbine and transformer lubrication. Crude oil residues of Middle East oil from ship cargo tanks were completely tolerated by both normal and eczematous skins. No instance of sensitization was recorded to the oxidation inhibitor additive (0.1% phenyl-α-naphthylamine) or to the copper-staining inhibitor.

Comparison of the two methods of patch testing indicated that in normal skin with all types of oils tested there was little difference between the number of positive reactions from the prophetic test and the insult test with the final 48-hour challenge. In eczematous skins, on the other hand, many more reactions occurred on the repeated insult test than on the subsequent 48-hour challenge, e.g., turbine-transformer oils 9:1, E.P. cutting oils 26:20, and soluble cutting oils 14:1.

On the average with all the oils, four to six insults were required to "overwhelm" the resistance in eczematous skins. One may assume that positive responses to uncompounded lubricating oils were primary irritant reactions. It is not easy, however, to interpret the results of insult tests with the compounded E.P. oils with respect to differentiating primary irritant from sensitization responses. There was no qualitative difference in the test insults from which to judge.

Presumably a positive 48-hour challenge should indicate a sensitization effect, but one cannot exclude this as being a primary irritant reaction. Most of the challenge positives occurred in eczematous skins; only one occurred in a normal skin. The majority of the challenge positives were provoked by the oil D, which was highly contaminated with metal.

A possible day to day variation in skin tolerance itself also introduces difficulty of interpretation, as was seen with the soluble cutting oil. Here several doubtful positive reactions either preceded a later definitely positive reaction or disappeared in spite of continued insults, only to be followed in time by later positive reactions. In some subjects after positive reactions were noted, the skin was allowed to rest and recover for 10 to 14 days before insults were started again on the same site. The insults were then tolerated for 6 to 10 additional occasions before the reappearance of the positive reactions.

Repeated insult patch testing is time-consuming and, judging from the results, a 48-hour patch test gives a good indication of any lowered tolerance to compounded E.P. oils. The repeated insult patch test also appears to have been helpful here in the case of the soluble cutting oils whose long exposure effects could not have been assessed by prophetic patch testing.

The high percentage of eczematous skins intolerant to spindle oil (for turbines and transformers) and to insoluble E.P. and soluble cutting oils, together with the irritant reactions of normal skins only to the used metal-contaminated sulfochlorinated E.P. oils or the soapy emulsion soluble oils, indicate the need for using good judgment in considering persons with such skins for jobs involving oil exposure. The advice usually given seems wise, that eczematous skins should not return to unlimited industrial contact with lubricating or cutting oils until the skin has become stable again. Early detection and treatment of dermatitis with due consideration as to the advisability of continued contact seems relevant. Because eczematous skins even when seemingly intact have a reduced tolerance, patients with convalescent skins after dermatitis from engineering oils should, on return to work, have only a limited and gradual exposure before re-

turning to full contact. That cutting oils should be clean and free from excessive metal contamination must be, as is known, an obvious prophylaxis.

Summary

1. Prophetic and repeated insult patch tests were performed on the intact skin of a group of normal and eczematous subjects with refined spindle lubricating oil, several types of E.P. cutting oils, and soluble "suds" oils.

2. Normal skins showed a complete tolerance to turbine and transformer oils and to clean used sulfurized and sulfochlorinated E.P. oils. Some irritant reactions, however, occurred from continued exposure to sulfochlorinated E.P. oils which were contaminated with metals and to soluble oils.

3. Intolerance to all tested oils was demonstrated in 30-36% of eczematous subjects with apparently intact skin.

4. Use alone (oxidation), in this instance, did not appear to influence the irritancy of the lubricating spindle oil but the irritant effect of E.P. oils was much increased by the degree of metallic contamination which resulted from use.

5. No sensitization was recorded to 0.1% phenyl-α-naphthylamine (antioxidant) or to copper-staining inhibitor additives.

6. Repeated insult patch tests produced more reactions in eczematous than in normal skins.

7. Irritant effects of lubricating and cutting oils are largely influenced by the presence of a completely intact epidermis and absence of eczematous skin changes. Reexposure to engineering oils after dermatitis is discussed.

Acknowledgment

I wish to thank Henry Prichard & Co. (1947) Limited, Bristol, for the supply and preparation of the oils and in particular Mr. R. Croft (Chief Chemist) for much helpful advice and for the technical information supplied in this paper.

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INDUSTRIAL TRAUMA

I. Principles of Early Management

James C. Drye, M.D., Louisville, Ky.

MORE USEFUL years of life are lost because of trauma than because of any disease, including heart disease, diabetes or cancer. Non-fatal injuries, in spite of our compensation laws, company sickness and accident policies, and so forth, all too frequently change the status of a man from that of a happy, useful, self-respecting contributor to society to that of a man who has become an embittered, helpless parasite. We must do our utmost to prevent trauma and, when it does occur, we must minimize its effect by skillful surgery and rehabilitation.

Trauma presents itself in never-ending variations, but still patterns of injury can be seen. An understanding of these injury patterns is helpful in predicting the nature and extent of the injury and its treatment. Thus, the combination of head injury, severe chest trauma, and extremity fractures in automobile accidents is well known.

In most modern industries, on the other hand, life-threatening accidents are far more unusual than those which may cause disability. The potentially crippling accidents which most frequently involve the peripheral area of the body—that is, fingers, hands, feet and toes, eyes, and head—tend to be peculiar to the machines of a particular industry. Examples are the corn-picker hand of the farmer, the heel fracture and tear of the Achilles tendon occurring when a dolly truck overruns a workman from the rear, the crushed hand of the press operator, and the severe crushing of the miner's leg from a mine coal car. Experienced industrial surgeons know these patterns and consequently know pretty well what to expect in the injuries that commonly occur in the particular industries they serve.

In this, the first of a series of articles on industrial trauma, it is my purpose to discuss the general early definitive management of the injured, particularly the severely injured. Subsequent articles will deal with specific injuries, both major and minor, and will give emphasis to the early management of the injured.

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Immediate Measures

The history of the mechanics of the accident, of the immediate effects of the accident on the patient, and a consideration of the patient's past medical history are all important. For example, the knowledge that a man has been pinned between the tailgate of a truck and a loading dock means but one thing to the experienced surgeon—exploratory laparotomy—as does the knowledge that a wheel of a truck or other heavy vehicle has passed over the abdomen. Similarly, other causes of injury will be known to the industrial surgeon to produce a fairly standard type of injury. A study and review of the records of the accidents of a given industry, with emphasis on the surgical implications of the resulting injuries, will help the surgeon develop this sort of understanding.

The history of the state of consciousness from the moment injury should be ascertained of all patients, keeping in mind the "lucid interval" of epidural hemorrhage. When spine injuries have caused paralysis, it is important to know whether the onset of this complication was immediate or delayed.

The length of the time interval between the occurrence of injury and hospital admission must be known. The interval may be so short that shock has not yet become apparent. An extremely long interval, on the other hand, may preclude primary closure of wounds and also necessitate changes in plans for the treatment of open fractures.

The past medical history of the injured patient may be of critical importance. Thus, the patient with preexisting cardiovascular disease, alcoholism, or diabetes will require special preparations for surgical procedures and treat-

ment. The industrial physician should be at an advantage in this, as this information should be in the health record of the injured employee. It would be well if a copy of this record were sent along with the patient to the hospital. If this cannot be done, the necessary information must be obtained from the patient himself or his relatives.

Good management of trauma frequently requires that the development of the patient's history, the special diagnostic procedures, physical examination, and treatment frequently overlap or be carried on concurrently without reference to an established sequence. In the process of doing these things it is essential that there be no unnecessary moving of the patient from stretchers to examining tables, to x-ray tables and operating tables, and so on. Arrangements must be such as to ensure that movement of the patient is kept at a minimum. Unnecessary and careless movement within the hospital may cause as much additional injury as is frequently caused in moving the patient from the scene of the accident to the hospital.

Severe respiratory distress has the highest priority for attention, even more so than hemorrhage. It usually is due to obstruction of the airway, an open pneumothorax, or a severe flail chest. The cause of the distress is generally obvious, and immediate attention should be given to it. Occasionally the trachea may be obstructed as a result of fractures or hemorrhage in the neck, and an immediate tracheostomy may be lifesaving.

If severe external hemorrhage is present, as evidenced by bloody clothing or blood on the stretcher, the site of the hemorrhage should be located immediately. The clothing should be cut away in order to avoid needless injury such as opening of closed fractures and so forth. When the site of hemorrhage is found, it is to be controlled by clamps, pressure, or other appropriate means.

Shock

An evaluation of the degree of shock should be made early in all patients. The manifestations of shock vary, depending on age, time elapsed since the accident, and the individual variations of the patient. No one of the commonly observed signs is reliable as an isolated observation. In fact, particularly in vigorous young people, few or no physical signs may be present, even when it is obvious from the extent of injury that shock must be present.

From experience we know that severe multiple injuries and some types of specific injury, such as open fractures of the femur and extensive fractures of the pelvis, uniformly cause shock. In patients so injured, vigorous antishock therapy should be instituted immediately, even in the absence of clinical signs of shock. Blood pressure, pulse rate, pulse quality, degree of vein filling, color and temperature of the skin, presence or absence of sweating, and mental reaction of the patient should all be considered in evaluating the degree of shock. Dependence on any one or two of these things may be quite misleading so far as degree, or even presence, of shock is concerned.

There is a tendency to be too dependent on blood pressure determinations in ascertaining the presence or the severity of shock. It is true, in studying large series of patients, that those with lower blood pressures have worse prognoses than do those with nearly normal pressures. However, in the individual patient the correlation is not always dependable. All surgeons have seen patients with nearly normal blood pressures who not only show many of the other signs of shock but obviously are in moderate or severe shock. The response of the blood pressure to treatment, on the other hand, is a dependable guide to the severity of the shock. If the blood pressure shows a good and sustained response following minimal amount of intravenous therapy, it is fairly certain that the degree of shock is not great. Further, so long as the blood pressure continues to rise with the treatment, one may be reasonably certain that the shock is being overcome, with exceptions only in certain types of head injury.

Pulse rates are extremely variable when shock is present and are more apt to be reflective of such factors as excitement and pain than variations in blood volume relationships. However, again, as with the blood pressure, a pulse which slows and improves in quality in response to treatment is usually indicative that the shock is being overcome. Generally speaking, the quality of the pulse parallels the relative normality of the blood pressure; but there are exceptions to this. The patient with a near-normal or even normal blood pressure, whose pulse feels thready and gives a light flick against the palpating finger, is usually in impending shock and should be treated as though in obvious shock. On the other hand, a patient who has a good full pulse and low blood pressure may be in better condition than the blood pressure reading by itself would seem to indicate.

Pallor, coolness, and sweating go hand in hand and indicate a high degree of compensatory peripheral vasoconstriction. These findings, associated with low blood pressure and

poor pulse quality, are diagnostic of the shock state. These skin signs may be the first to show improvement after treatment has been started.

The peripheral veins in their normal state are generally easily observed, and their state of filling is a valuable index. They are, of course, collapsed in shock. After treatment has begun, the state of filling can be valuable as a guide to further therapy. So long as the superficial veins remain empty, there should be no hesitation in giving blood rapidly; as they begin to fill, it is time for the blood infusion to be slowed, since this is evidence that the vascular bed and blood volume are coming into balance. If other signs of shock continue to be in evidence, the difficulty is not attributable to a discrepancy between the blood volume and the size of the vascular tree.

The usual laboratory tests, such as blood cell counts and hemoglobin determinations, are of little if any value in estimating acute traumatic shock. There are so many unmeasurable variables operating in the transfer of fluid across the barriers between the various fluid compartments, which, in turn, change the concentration of these elements in the plasma, that such findings mean little.

Blood volume determinations have found little use in cases of acute traumatic shock. Such studies are still cumbersome, difficult to perform, and time-consuming. Further, blood volume may vary in otherwise apparently similar individuals by as much as 500 to 700 ml. In addition, it has not been proved that the normal blood volume of a healthy person is necessarily the optimum blood volume for that same individual after he has been severely injured. An evaluation of the degree of shock and an estimation of the amount of blood to be given are to be based upon the clinical response as revealed by changes in signs and symptoms rather than upon laboratory tests.

Some patients do not respond even to the most vigorous antishock therapeutic measures. In a few cases the patients have sustained injuries of such severity that they are mortally wounded and nothing will revive them. However, one should never accept this as a true evaluation but should utilize every resource available to resuscitate a seemingly hopeless patient. Occasionally such a patient is saved by proper and vigorous treatment.

If the patient does not respond as expected to therapy, a search must be made to determine the cause for the lack of response, and reexamination of the patient is indicated. At times, operative intervention is necessary to correct the cause of this nonresponsive shock. Bleeding, external or internal, from large vessels must be stopped by ligature or clamp. Unsplinted major fractures can cause shock to be resistant to treatment, since there is increased blood loss into the tissue upon every slight motion. It is futile to force blood into a patient through an 18-gauge needle only to have it escape through a 12-gauge-size blood vessel. Another cause is massive peritoneal soiling, as from a torn right colon. Such patients will remain in shock despite the most vigorous antishock treatment until the abdomen has been cleaned out.

A common cause of treatment failure is underestimating the amount of blood loss, particularly in connection with extensive fractures about the pelvis and fractures of the spine. Such injuries may lead to enormous blood losses as retroperitoneal hemorrhage. Retroperitoneal hemorrhage of this type is most frequently from the veins. It is best left alone unless it can be demonstrated that arterial bleeding is an important factor in the problem. These massive hemorrhages require massive blood replacement.

Examination of the Patient

The examination of the patient proceeds as the shock is evaluated and therapy started. Examination must be complete and thorough, inasmuch as an obvious or painful injury frequently masks a less evident and sometimes more lethal injury. All clothing must be removed from the patient. However, before this is done, a cursory examination should be made to locate any major fractures or other serious injury. The head may be inspected and felt, looking into all orifices for blood and cerebrospinal fluid. The neck should be examined for contusions, lacerations and tenderness. If there is tenderness over the cervical spine, or pain on the slightest motion, the area must be protected until x-ray films have been made.

The clavicles and chest may then be subjected to pressure and compression, beginning lightly and becoming firmer. Fractures will, of course, cause pain. Flail chest may be seen and felt. Next, the pelvis should be subjected to pressure and compression over the pubis and iliac spine, with slight pressure at first, and then firmer pressure if no injury is found. In a similar manner the upper and lower extremities should be completely checked.

The clothing should then be entirely removed. If the preliminary examination has revealed injuries which can be aggravated by motion, the clothing should be cut away. The destruc-

tion of a hundred-dollar sport coat is a small price for the prevention of conversion of a closed fracture into an open one.

After the clothing has been removed the same maneuvers should be applied as were carried out in the initial examination, and a more thorough examination done. The entire body surface should be inspected for the presence of abrasions or contusions, which may point to deeper and more serious injuries. One should again search for any signs of external bleeding, and then proceed in an orderly manner to examine the patient completely, evaluating the injuries as the examination progresses.

Head Injuries

The most important indicators of the severity of brain damage are the degree and the duration of unconsciousness. The deeper and the longer the state of unconsciousness, the graver the prognosis. Accurate notes should be made as to the state of consciousness. Convenient terms which can be used are "coma," "stupor," and "restlessness." The state of consciousness should be recorded in these terms at definite intervals. Rarely the skull may be opened by some freak occurrence, with the brain protruding through this opening, and the patient still be conscious. However, this will be obvious.

Localizing signs such as dilated pupils, decreased deep tendon reflexes, and the abnormal Babinski reflex, should be sought for and the results recorded, along with a statement as to the state of consciousness, to be used as a future guide in the administration of treatment.

The presence or absence of depressed skull fracture should be determined. If the scalp has been split, the skull may be explored gently with a sterile hemostat to search for such a depression. All open, depressed fractures over the dural sinuses and over the nonsilent areas should be attended to fairly soon. Closed, depressed fractures over silent areas may be left alone for as long as several days, or until the patient is in suitable condition for major surgery.

The nostrils should be carefully inspected. The presence of cerebrospinal fluid means a fracture through the ethmoid or frontal sinuses. The presence of blood in the ears which cannot be attributed to an outside source indicates a basilar skull fracture and is a bad prognostic sign.

The possibility of epidural hemorrhage must be kept in mind at all times. Without proper treatment such injuries are lethal; but when they are discovered early and properly treated, the outlook becomes good. Evidence of trauma over the temporal areas should always bring the possibility of epidural hemorrhage to mind.

As a rule, roentgenography of the skull should be deferred. The amount of brain damage should be the deciding factor, and not the fracture of the skull, unless the fracture is depressed. Occasionally, films are immediately necessary to determine the presence or absence of a depressed fracture. It cannot be emphasized too strongly that in all cases of brain damage serial determinations of pulse, blood pressure, state of consciousness and the usual localizing signs should be made and carefully recorded.

If evidence of damage to the neck was found during the preliminary examination, further study, as needed, can now be done.

Chest Injuries

The chest next is carefully examined. Inequality of excursions or paradoxical movements denoting flail chest may be seen. Fractured ribs are determined by a point of tenderness and noted for intercostal nerve block.

Evidence of subcutaneous emphysema should be sought for over the chest wall and the neck. Crepitation may be felt. If it is most prominent over the base of the neck or the suprasternal notch, mediastinal emphysema is likely to be present. Crepitation of the skin and subcutaneous tissue usually means a fractured rib and torn lung. It may be due to a tear of the bronchus or bronchiole, with no tear in the visceral pleura. The air, in this case, escapes along the course of the bronchi and blood vessels into the mediastinum, and then out over the chest and neck. In the presence of emphysema one should be doubly on guard for tension pneumothorax.

The usual physical signs of fluid in the thorax are sought. Needle aspiration is valuable in the diagnosis of blood or air in the pleural space. If any is found, it usually is wise to continue aspiration until the space is empty, for therapeutic reasons as well as to determine whether there is active bleeding or continued leak of air into the pleural space. An intercostal drainage tube, connected to a water trap or to suction, may become necessary. The return of respiratory function to normal has a high priority in treatment, since anoxia increases shock and is particularly harmful to patients with head injuries. Immediate steps should be taken to restore this function to normal.

Severe chest injuries produced by automobile accidents typically consist of fractured ribs,

hemopneumothorax, and the "wet lung syndrome." A dramatic change for the better can be produced in these patients by aspirating the airway, blocking the intercostal nerves, and emptying the pleural space by aspiration. Flail chest may be helped temporarily by simply placing a small sand bag over the area. Towel-clip traction should be started as soon as possible. By using one to three towel clips, fastened about the ribs in the involved area, and attaching their handles to light overhead, balanced traction, most flail chests can be stabilized. Within a few days the chest regains sufficient stability so that the clips may be removed.

Extremities

Examination of the extremities should be thorough and complete. Mild cursory examination will, of course, reveal major fractures, but many permanent and crippling injuries are all too easily overlooked. The loss of function in a hand may convert a skilled workman into an economic parasite.

All extremities should first be inspected for lacerations, contusions and abnormal contours or positions. Next, they are gently, and then more firmly, palpated. If no evidence of fracture is found, they then may be moved slightly and gently at first, and then more extensively if no injury is demonstrable.

In all cases of extremity injury the peripheral nerve and vascular function must be determined and recorded. The proper time to find a wrist drop due to radial nerve injury is at the time of injury, not when the cast which was applied for the fractured humerus is removed. Peripheral nerve injuries usually can be sufficiently delineated by considering the areas of anesthesia and a few simple tests for motor function. A safety pin is a convenient instrument to use for determining sensation, using the sharp and dull ends as indicated. Areas of anesthesia may be mapped out. Tests for motor function, under these conditions, need not be unduly complicated. Every surgeon should learn a few simple maneuvers to demonstrate whether the major nerves are intact or injured.

If the wrist can be dorsiflexed, the radial nerve is intact. If the tips of the thumb and fifth finger can be apposed normally and the second and third fingers strongly flexed, the median nerve is intact. If while in extension the fingers can readily be abducted and adducted, the ulnar nerve is usually intact.

Active contraction of the quadriceps muscle or the ability to extend the leg at the knee indicates an intact femoral nerve. If the thigh can be adducted, the obturator nerve is intact. If the patient is able to flex the knee and move the foot, the sciatic nerve is intact. The ability to dorsiflex the foot indicates an intact common peroneal nerve. To test the deep branch of the peroneal ask the patient to invert the foot. Eversion of the foot indicates that the superficial peroneal nerve is intact. The ability to flex the toes and plantar flex the foot indicates that the tibial nerve is intact.

The state of the circulation must be determined and recorded in all extremity wounds. The presence or absence of the peripheral pulse and the temperature and color of the skin should be observed.

Intra-Abdominal Injuries

Intra-abdominal injuries are treacherous. Even in cases of badly damaged viscera with soiling, there may be a lack of physical signs because of shock, prostration, or more painful injuries in other areas. One should look for any signs of skin damage such as abrasions and contusions over the abdominal wall, because these may point to deeper injury. One should watch for the usual signs of peritoneal irritation. It must be remembered that fractures of the lower ribs will lead to upper abdominal signs. An increased area of percussion dullness which is nonshifting and located over the spleen suggests splenic rupture. If this is accompanied by an otherwise unexplainable pain in the left shoulder, it is highly diagnostic. From experience we have found this to be quite reliable.

Needle aspiration in the four abdominal quadrants through an 18- or 20-gauge needle is helpful if positive, but should be disregarded if negative.

Subcutaneous emphysema over the abdomen is rare. When present early, it may be due to air trapped in the layers of a deep lacerating wound. If it develops after a few hours, it usually means a tear in some retroperitoneal portion of the intestine.

Gastric suction through a nasogastric tube is helpful. It is of the utmost importance that all patients who are to be anesthetized have their stomachs emptied. Vomiting and aspiration during emergency operations cause one or more deaths per year in most large hospitals. Further, if blood is aspirated from the stomach, it is diagnostic of stomach injury, provided sources from above can be ruled out.

Genitourinary Injuries

All patients considered to have possible genitourinary injuries should be catheterized,

and an examination of the urine specimen should be made. Gross blood usually means bladder or urethral damage. Microscopic blood usually means kidney damage.

If blood is found in the bladder, the bladder should be irrigated with sterile saline solution. The amount of fluid injected should be measured; a deficiency in amount of fluid returned indicates a leak in the bladder. Cystography should be performed in any doubtful case, for the more reliable information it can give.

If the catheter cannot be passed, it generally means a torn urethra. A suprapubic cystotomy and splinting of the urethra will be required. If the bladder or urethra is found to be damaged, a search for extravasation of urine should be made about the genitalia and lower abdominal wall. The urine also should be subjected to the usual laboratory analysis, since preexisting diabetes or urinary tract disease may be present in those patients whose previous histories are unknown.

Digital rectal examination is especially indicated in patients with crushed pelves or with possible intra-abdominal injuries. Fresh blood on the examining glove may indicate a torn rectum or colon. Occasionally, a tear may be seen through a proctoscope or sigmoidoscope.

Discussion and Summary

The variations of trauma are almost endless, the forces exerted can be great, and multiple injuries of varying degrees of severity are the rule. The physician who attends these victims is called upon to meet the most demanding requirements of thoroughness, common sense, judgment, and decisiveness of action if the patient is to do well. In a presentation of this sort some order of procedure must be used, but the multiple injuries do not lend themselves to any preconceived order. First things must come first, and the physician must use judgment as to the relative priority of such multiple injuries. Thus, generally speaking, respiratory difficulties have the highest priority for treatment, but it would be foolish indeed to spend 20 minutes aspirating a mild pneumothorax while the patient was bleeding from a large, open, accessible vessel. Contrariwise, it would be equally foolish to worry about a bleeding artery in a patient with complete obstruction of the airway.

As examination and treatment proceed, governed by priority of the severity of the various injuries, it must be remembered that finally a complete, orderly examination of the entire patient must be done. The end of the examination and final evaluation of the patient can only come after all injuries have been identified and their severity accurately assessed.

Although the vast majority of industrial accidents result only in minor injuries, too many still result in permanent disability and death. The very best surgical care should be provided to these accident victims, for truly they are our most pressing problem in terms of loss of useful and productive years of life.

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The theme of the conference will be "New Views on Employee Health and Labor Management Objectives." The views to be expressed by speakers representing labor, management and the medical profession will touch upon current practices, deficiencies, and discriminatory practices as they exist today in occupational health services. Views on health progress in an industrial society, mental health aspects, and ideals to strive for in the future also will be presented by means of case studies, orientation lectures, and general evaluation discussions. For further information, write Greater Vancouver Health League, 1300 Robson Street, Vancouver 5, B.C., Canada.

Penetrating Foreign Body in the Liver

Joseph B. Tauber, M.D., Dr. P.H., Aliquippa, Pa.

A LIVER injury occurring in industry is usually the result of a severe contusion of the abdomen by a heavy crushing force. Injury to the liver by penetrating foreign body with a comparatively small portal of entry and without extensive but with significant parenchymal damage is commonly caused by high velocity foreign bodies like shell fragments. Though this type of injury is most unusual in industry we are able to report such a case.

The accident occurred in the tin plate department of a steel mill on June 1, 1957. A coil of strip was being threaded through a five-stand tandem mill, when a pistol-shot-like noise was heard and the employee next to the mill felt a pain in the right side. He and other workers around him noticed that he was bleeding from the side. He was taken to the plant dispensary.

The staff physician's examination revealed a 34-year-old, moderately obese, white man on the examining table, in good spirits, chatting away in a loud voice with the nurse and with his foreman, who accompanied him. He explained before the examination that he felt no pain, that he felt perfectly well, and that he did not want "to waste too much time here." In fact, he asked "to put a stitch into the small wound, or just bandage it" so that he could return to work.

The skin wound was 1½ to 2 inches long and evidently deep, at the ninth right intercostal space in the anterior axillary line. It had sharp, clean, and slightly gaping edges, so that the yellow subcutaneous fat-layer was seen with a very moderate amount of bleeding of dark venous blood. The patient's principal concern was the inconvenience he caused the medical staff, and the fact that being in the dispensary he could not perform his work. His temperature was found normal, his pulse of good volume and regular and the rate was 76 per minute. His skin was dry and of good color; his cheeks were rosy. The abdomen was soft and nontender. The chest was clear to percussion and to auscultation. A diagnosis of nonpenetrating skin laceration compatible with these signs and symptoms, is supported by a report of the preliminary, rapid investigation of the accident sent in from the plant. This stated that the injury was caused by the edge of the undamaged tin plate, hitting and cutting the body of the injured. No part of the plate, nor of the machinery was missing.

Dr. Tauber is Plant Medical Director, Jones and Laughlin Steel Corporation.

While the patient was quiet there was only minimal or no bleeding from the wound. He moved, however, not infrequently, and at some of these movements 10-15 ml of dark, venous blood rushed forth from the wound, suggesting contact with a cavity. He had several consecutive bowel movements in quick succession, with bulky, soft, but not watery stools of normal color, without any blood. At this time he complained of some epigastric discomfort and nausea. He did not vomit. He explained that all this resulted from the "good mouthful of chewing tobacco I swallowed when I was hit."

About 40 minutes after the beginning of the examination the patient's pulse became weak and rapid, and he became pale and clammy. Nikethamide (Coramine), 2 ml, and 1 pint of 5% dextrose in Ringer's solution were administered intravenously. In about 20 minutes his pulse was of good volume again, and around 78-80 a minute. He felt well again; his skin became dry; and the pink color of his cheeks returned. Auscultation and percussion of the chest remained normal the whole time. The abdomen felt soft and nontender. The patient was transferred by ambulance, on stretcher, to the plant health center for radiography of the chest, as right hemothorax was suspected.

During roentgenography the patient started to go into shock again, and i.v. drip infusion was started. The roentgenogram revealed a quarter-sized metallic foreign body with ragged edges under the vault of the right diaphragm. A lateral view taken revealed it to be lodged deep in the liver.

The patient was transferred to the hospital, and laparotomy was performed. The foreign body was removed from the liver by a right upper quadrant incision and via the portal of entry in the liver. It was found to have penetrated the chest wall, the right costophrenic angle, and the diaphragm, and to have passed into the liver. Internal bleeding was caused from severed diaphragmatic vessels. He had a stormy postoperative course because of impending hepatic failure, but he responded well to blood transfusions, cortisone, and other supportive measures. He later developed a subdiaphragmatic abscess which had to be opened, after which he had a discharging sinus for about three months.

The patient was absent from work for four months, was on light duty for the following three weeks, and then went back to his regular job. He has been working regularly ever since, and is doing well.

Editorials

WITH THIS issue we begin our second year of publication of the JOURNAL OF OCCUPATIONAL MEDICINE. Our birthday party is a special issue that deals with some of the fundamental problems of a major segment of industrial medicine: occupational skin diseases.

Plans for the party began early last spring. Our friend, Dr. Raymond R. Suskind, took on the job of designing the issue, inviting the contributors, and bringing the total project to completion. To Dr. Suskind and his colleagues in this country and in Great Britain go our sincere thanks.

Some Things New

GROWTH and change are the marks of life. Volume 2 of our journal begins with two additional features: an abstracts section dealing with environmental physiology and a section on case reports. By environmental physiology we mean the interactions of the working subject and his environment. This section will begin in a modest way. The literature on physiologic responses to thermal stress will be abstracted by Capt. David Minard, and the literature that deals with responses to changes in atmospheric pressure will be covered by Capt. Harry J. Alvis. Both men are officers of the U.S. Navy, respectively assigned to the National Naval Medical Center and the Pacific Fleet.

Reports of cases of occupational illness or injury are expected from physicians in industrial practice everywhere. The invitation for wide participation in this type of contribution was issued earlier by DR. D. JOHN LAUER, the Association's President. We repeat it here: case reports are wanted from the medical officers of a wide variety of industries. The first one, by Dr. Joseph B. Tauber, in this issue deals with a penetrating foreign body in the liver.

Another change, and one that will be welcomed by a large number of our readers, is a better coverage of surgery topics than was possible during our first year. We begin with a review entitled, "Industrial Trauma—Principles of Early Management" by DR. JAMES C. DRYE of the University of Louisville.

Dr. Edward V. Henson, a member of our Editorial Board since its beginning, has been promoted to Assistant Editor. In this capacity he will manage the Abstracts Section of the Journal—a function he has carried on without recognition for the past six months. The vacancy on the Editorial Board created by this change has been filled by the appointment of Dr. Robert C. Combs. More details about these gentlemen will be found in the People and Events columns of this issue.

Binding Time

THE BEGINNING of another new year is the time when most of us have to face up to the question as to which periodicals we will keep around the office and which ones must be thrown away because of shelf space limitations. Those which are to be kept get sent to the binder's, and those we unquestionably don't want to see again get pitched in the general direction of a waste basket. A large number of gray-zone items, about whose permanent value there is a stubborn ambivalence, are sent to some vague place called the library.

If all this sorting, sifting, and classifying produces unneeded copies of the early issues of the JOURNAL OF OCCUPATIONAL MEDICINE these should be sent to our publication office in Chicago. We continue to get orders for full sets of Volume 1. Such orders are filled for medical libraries, medical societies, and technical publications; but we cannot supply copies of early issues to individuals. Our embarrassment in this regard can be relieved if I.M.A. members will send us all the copies of the January, February, and March, 1959, issues they can spare.

Book Reviews

SYMPOSIUM ON INSTRUMENTATION IN ATMOSPHERIC ANALYSIS. American Society for Testing Materials Special Tech. Bulletin No. 250. Price, $2.25 (ASTM members, $1.80). Pp. 64. American Society for Testing Materials, 1916 Race St., Philadelphia, Pa., 1959.

This little booklet presents the four papers which were read in Boston, Massachusetts, June 23, 1958. The symposium dealt with the activities and accomplishments of the Subcommittee on Instrumentation of ASTM Committee D-22, on Methods of Atmospheric Sampling and Analysis.

The first paper describes a particle counter that is able to count and classify individual particles greater than ¼ micron in diameter when present in an extremely dilute atmospheric aerosol. The second paper reviews the instruments useful for the continuous evaluation of airborne particulates by filtration through fibrous media and attempts to relate absorbance to mass concentration, carbon content and to the number and size of the collected particles. The third paper compares five instruments useful in air pollution work. Included in the discussion are chemical recorders for determining concentrations of oxidant and nitrogen dioxide, two spectrometric instruments for monitoring the same pollutants, and gas chromotography. The last paper discusses the background of the Adak and SRI instruments which were developed for the determination of fluoride in the air and gives a detailed description of the SRI instrument which can detect from 0.2 to 10 ppb fluoride in 10 to 20 minutes or less.

The papers present a great deal of background information in one of the areas of activity of ASTM Committee D-22 and will be helpful for understanding the tentative standards which the committee is recommending to ASTM. The papers recount some of the successes of the committee in evaluating the instrumental methods in current use for the continuous monitoring of pollutants present in the air in extremely low concentration.

—J. CHOLAK, CH.E.

TWENTY THOUSAND NURSES TELL THEIR STORY: A REPORT ON THE STUDIES OF NURSING FUNCTIONS SPONSORED BY THE AMERICAN NURSES' ASSOCIATION. Everett C. Hughes, Ph.D.; Helen MacGill Hughes, Ph.D.; and Irwin Deutscher, M.A., M.S., With a Foreword by Agnes Ohlson. Price, $5.75. Pp. 280. J. B. Lippincott Company, E. Washington Square, Philadelphia 5, Pa., 1958.

The book presents in a most readable form a synthesis of the data of 34 nursing studies. All focused on the nurse, who she is, what she does, where she works, her education, and her relationship with coworkers and patients. The authors, who are sociologists, not nurses, wisely caution the reader of the hazards involved in making broad generalizations. They point out that this book can be only as valid as the studies from which the data presented are drawn.

Two studies about the industrial nurses, Wendell Smith's "The Industrial Nurse," an analysis of her functions and Erna Barschok's "Today's Industrial Nurse and Her Job" are included. These studies, like some of the others reported, ask many more questions than they answer.

The book is stimulating and thought-provoking. It is also disturbing. It points up where we are in nursing and indicates in all too vivid terms that we have a long way to go.

—MARY LOUISE BROWN, R.N., M. LITT.

ELEMENTS OF BIOPHYSICS. James E. Randall. Price, $8.00. Pp. 331, with Index. Year Book Publishers, Inc., 200 E. Illinois St., Chicago 11.

The reviewer can do no better than to quote the author's own preface, in which he says: "It is important for the reader of this book to understand that this text was not undertaken in an attempt to define what should be considered as biophysics, but rather as an effort to present to the student of biology and medicine the physical and quantitative concepts of basic importance to these fields."

If the limits are recognized, the prospective reader will not be disappointed but will find a well-written elementary text of value to the medical practitioner of practical as well as of academic bent.

There is a continuing need for quantitation in biological thinking and experimentation. The day is over of acceptance of casual morphologic description without quantitation. Gone, too, are the days when the physician could content himself with what he had learned at school and retire into active practice without the necessity for further study. Dr. Randall's discussions of mathematical concepts are intended for the reader without mathematical training. He performs admirably. The jargon of modern medicine and modern research and industry becomes intelligible in his descriptions of Error, Accuracy, Precision, Instrumentation, and Feedback systems. The treatment of Statistics is perhaps briefer than we are accustomed to in even an elementary text, but in context it appears adequate and does not suffer from the usual defect of attempting to make inadequate statisticians of adequate biologists.

Chapters on electricity and electromagnetic energy are included. The physician who wishes to gain an acquaintanship with the area and perhaps a lead to further reading would be well advised to start with Dr. Randall's book. It grew out of a series of lectures for medical students and is well suited as an introductory text.

—MITCHELL R. ZAVON, M.D.

Abstracts of Current Literature

Internal Medicine

Pleural Biopsy in the Diagnosis of Pleural Effusion: A Report of 200 Cases. P. Mestitz,* M. J. Purvas, and A. C. Pollard. Lancet 2:1349 (Dec. 27) 1958. (*Middlesex Hospital, London, W. I.)

Patients who had had pleural biopsies were divided into three groups: (I) those who had a pleural effusion and the object was to determine the cause; (II) the pleural effusion was not the foremost problem; and (III) patients with residual pleural thickening with solid lesions in or near the pleura, or with no radiological evidence of pleural disease.

One advantage of pleural biopsy is early correction of a wrong clinical diagnosis. To illustrate: A patient 51 years of age, a heavy smoker with a history of weight loss, increasing dyspnea and a massive pleural effusion, was diagnosed as having inoperable bronchogenic carcinoma. The pleural biopsy revealed tuberculous granulation. Chemotherapy resulted in recovery.

Negative biopsies are unreliable, especially in case of malignant intrathoracic growths. Pleural plaques are easily missed. False negative results occurred in about 50% of patients with intrathoracic malignant disease. In tuberculous effusions, however, the biopsy will be positive four times out of five.

The authors conclude that pleural biopsy is simple and safe. It is a most reliable diagnostic technique when tuberculosis is suspected. The diagnosis can be established in about 80% of tuberculous effusions and in about 60% of malignant effusions. Every patient with a pleural effusion of questionable etiology should have the benefit of pleural biopsy. —S.H.S.

Aspirin and Gastric Hemorrhage. A. Muir, and I. A. Cossar. Lancet 1:539 (March 14) 1959.

The authors undertook a recent investigation to determine whether aspirin can produce gastric hemorrhage. About 50% of a series of patients with gastrointestinal bleeding had taken aspirin within 48 hours of their initial hemorrhage. Only 15% of the control series gave such a history. Of the patients with gastrointestinal bleeding following the ingestion of aspirin, about 50% knew that aspirin gave them indigestion. Aspirin is apparently more dangerous if taken on an empty stomach. It is believed that aspirin is an irritant, and that crushing the tablet has no effect in diminishing its irritant property. The authors feel that an acute erosive gastritis results.

This is the second investigative series the authors have undertaken on this problem, and they are convinced that aspirin is a dangerous drug, especially in patients in whom it causes indigestion, and in those with peptic ulcer. Their investigation suggests that one gastroduodenal hemorrhage in eight is caused by aspirin, and they urge a much more detailed study of its effects on the stomach. —S.H.S.

Effects of Periodic Mental Stress on Serum Cholesterol Levels. S. M. Grundy,* and A. C. Griffin. Circulation 19:496 (April) 1959. (*Clayton Foundation for Research, Houston, Texas)

Emotional stress has been implicated by several workers as a causal factor in atherosclerosis by alteration in cholesterol metabolism. The present study reports the relationship between fasting blood cholesterol levels in medical students during two midquarter observations and two quarter examination periods. Fifty medical students showed a rise of 16.5% in their blood cholesterol levels during their winter quarter examination period, compared to their winter quarter control period. Forty-seven of the same group showed a similar average change of 11% during the spring examinations compared to the spring quarter control observations. The authors suggest that endocrine imbalance during stress must be considered as a possible cause of changes in serum cholesterol. —J.J.T.

Joint Responsibilty (Editorial). W. B. Bean. A.M.A. Arch. Internal Medicine 103:681 (May) 1959. (*University of Iowa, Iowa City)

There can be no doubt that modern medicine is much indebted to the pharmaceutical industry. Theoretical considerations based upon practical experience have led to the design and synthesis of many new compounds of therapeutic value. Standards of purity and limits of acute toxicity have been rigorously observed. Somewhat more difficult is the regulation and control of the purported efficiency of drugs. Similarly, safeguards against cumulative ill effects, intolerance to prolonged therapy, damage to the liver or hematopoietic system, and the like are inadequate. Many, perhaps most, pharmaceutical companies exercise scrupulous caution in releasing new drugs. A few, unfortunately, seem to find some conflict between assuring a profit for the stockholder and the strictly moral and professional aspects of medicine. Trade secrets, the need for rapid action in the market, advertising techniques, and other considerations make any efforts to deal constructively with the problem difficult.

A plea is made for responsible persons in medicine, industry, and government to face existing problems in the interest of the public. —L.W.

Blood Cholesterol and Uric Acid of Healthy Medical Students Under the Stress of an Examination. F. Dreyfuss,* and J. W. Czaczkes. A.M.A. Arch. Int. Med. 103:708 (May) 1959. (*The Hebrew University Hadassah Medical School, Jerusalem)

The blood cholesterol, alpha- and beta-lipoproteins, and uric acid levels were determined in a group of 21 medical students immediately before

an oral examination and 48 hours later. On the first determination of cholesterol under stress the level was elevated in 20 of 21 instances but the elevation was within the normal range for the method used. The "increased" cholesterol seemed to be associated with the alpha-fraction, but results were inconclusive. The uric acid levels were unchanged. —L.W.

Medical Care Administration

Foundation for Medical Care. D. C. Harrington.* J.A.M.A. 170:963 (June 20) 1959. (*President, Foundation for Medical Care of San Joaquin County, Stockton, Calif.)

The Foundation for Medical Care of San Joaquin County was set up in 1954 for the purpose of assuring the availability of medical care to all people within the jurisdiction of the San Joaquin County Medical Society. It was designed especially to bring medical care within the reach of the population regardless of their ability to pay. At the present time 97% of the active doctors of the medical society are members of the Foundation. The goals of the Foundation are (1) a group payment program which the Foundation could sponsor and endorse, (2) an individual and family prepayment program which they could sponsor and endorse, and (3) some method of assisting those not in a position to have group or individual prepayment programs.

The Foundation has (1) set up a fee schedule based on the California Medical Association Relative Value Study: (2) stimulated acceptable group insurance programs by six insurance companies and the California Physicians' Service: (3) stimulated a family and invidual contract offered by California Physicians' service: (4) embarked on a service contract with labor unions: (5) set up an advisory committee of interested citizens. In the face of ever-increasing coverage of prepaid medical care insurance, it behooves organized medicine to actively work with insurance companies, the Blue Shield Plans, and the consumer. —A.B.

The Case for Unionization of Hospital Workers. Nelson H. Cruikshank.* Mod. Hosp. 93:71 (July) 1959. (*Director of the Department of Social Security for the AFL-CIO, Washington, D. C.)

The decision as to whether or not hospital employees should be represented by unions in collective bargaining is up to the workers themselves, not the hospital administrators. Once the workers make that decision the conscientious administrator has no honorable alternative but to bargain in good faith. There is no law that requires a private, nonprofit hospital to engage in collective bargaining, but neither is there a law that forbids it.

Collective bargaining will not solve all of a hospital's personnel problems; however, responsible unions provide a safety valve which helps to settle legitimate grievances promptly, weed out the irrational gripes, and clear up honest misunderstandings.

Union organization will increase wages and consequently cost of hospital care. Some wage increases are inevitable, union or no union. The hospital employee has apparently rebelled at his traditional role of subsidizing hospitals. If the cost of running a hospital goes up because it starts to pay decent wages, this is one of the facts of life—both for the administrator and for the community.

The emotional heart of the argument against union recognition lies in the belief that this will somehow make strikes against hospitals "inevitable." The opposite is true, because in the absence of unions there will be no machinery for workers to bring their problems and proposals to management through their chosen representatives.

To tell today's hospital worker that he must be denied the opportunity to bargain collectively for economic and social advantages is to inform him that he is a second class—although dedicated—citizen. —A.B.

The Challenge of Occupational Health. A. J. Hayes.* Indust. Med. & Surg. 28:313 (July) 1959. (*President, International Association of Machinists, Washington, D. C.)

American workers lose more than 500,000,000 days of work a year as a result of illness. It is estimated that about 10% of this loss, some 50,000,000 days a year, is attributable to illness of occupational origin and that the wage loss due to occupational illness is in the neighborhood of $420,000,000 each year. These estimates are, in the eyes of the author, a vast understatement of the problem. There is no accurate record of the real toll of occupational diseases, and the concept of occupational disease is still woefully limited because of imperfect knowledge and incomplete research. Since 2 states have no occupational disease laws and 21 have laws covering only specific diseases, hundreds of thousands of cases of occupational diseases occur every year without being recorded. Our knowledge of occupational disease is imperfect and is growing more so owing to the introduction of new chemicals, materials, substances and processes into industry.

We can no longer afford the luxury of ignorance on the subject of occupational illness. While most of the hazards of today's industry affect only the worker directly exposed, in the industry of the future electromagnetic radiation may possibly affect generations yet unborn in the families of exposed workers. We are entering into the atomic age without having solved the health problems of the machine age.

Everything possible should be done to secure the most widespread dissemination of material developed in the field of occupational health. Organized labor will contribute in every way possible toward enlightening employers and workers alike to health hazards and methods of eliminating them. —A.B.

Abstracts

Mental Health

Use and Overuse of Tranquilizers. F. Orland.* J.A.M.A. 171:633 (Oct. 1) 1959. (*Department of Psychiatry, University of Pennsylvania School of Medicine, Philadelphia)

In the past 10 years there have emerged many psychochemotherapeutic agents, including reserpine, meprobamate, chlorpromazine, and promazine. Unlike the barbiturates, these drugs do not reduce cerebral oxygen consumption and presumably they act at subcortical levels. These drugs are now widely used by the general practitioner, who "renders an incalculable service as the first line of defense in controlling and managing emotional illness. Without his help, psychiatrists would be overwhelmed and inundated by patients who either do not immediately require psychiatric care or would not benefit from it."

The tranquilizing drugs do indeed provide a temporary relief, but they cannot change the basic personality structure and the administration of these drugs should be accompanied by psychotherapy. The prolonged use of the tranquilizers is not without certain disadvantages. "A certain minimum level of anxiety is a prerequisite for psychobiological survival, since it makes one aware of existing problems and furnishes motivation to seek their solution." There is the possibility, then, that the patient may become indifferent to his problems and to the realities of life if he relies on tranquilizing drugs. Moreover, a long-continued administration of tranquilizers may foster an undue dependency on them and upon the physician. The patient becomes more passive and helpless, leading to a perpetuation rather than a solution of his conflicts. —G.C.T.

Psychotherapy of Alcoholism. F. Lemere.* J.A.M.A. 171:266 (Sept. 19) 1959. (*University of Washington School of Medicine, and the Shadel Hospital, Department of Psychiatry, Seattle)

The recognition of alcoholism as a disease by the American Medical Association has put more responsibility on the physicians to treat patients with this condition. Alcoholism is now considered to be "our fourth most serious public health problem, being outranked only by mental illness, heart disease, and cancer."

The physician should not harbor any feelings of contempt or superiority or criticism, since the patient will already have encountered these previously and since they are of no therapeutic value. Theoretically, psychotherapy is the answer to alcoholism, but the initial step must be a recognition on the part of the patient that he has a problem. If the patient denies this, all the physician can do is to help him as regards his immediate state. The physician should not put any pressure on the patient to stop drinking unless there are present complications such as cirrhosis or delirium. In addition to psychotherapy, the author recommends various other resources such as Antabuse, Alcoholics Anonymous, and ministerial help. To sum up, the patient must accept the fact that he has a drinking problem and be educated to the necessity for total abstinence, and the therapist must have a noncritical and helpful attitude toward the patient. —G.C.T.

The Role of the Psychiatrist in a Prepaid Group Medical Program. L. L. Tureen.* Am. J. Pub. Health 49:1373 (Oct.) 1959. (*457 North Kingshighway Blvd., St. Louis 8)

The author, who is with the Department of Neurology and Psychiatry, St. Louis University School of Medicine, describes the functioning of the St. Louis Labor Health Institute, which provides medical care through a voluntary prepaid medical plan serving 15,000 eligible members. This involves approximately 50 physicians and dentists employed on a part-time basis. The Institute provides an integrated program of medical care, including hospitalization. Three psychiatrists provide 20 hours a week to the neuropsychiatric section.

In 1958, there were approximately 6,000 union members who, with their families comprise the 15,000 persons entitled to the Institute's care. The incidence of emotional disorders in this group appears to be around 6.6%, but this is probably a base line and 10% would be a more accurate estimate.

Tureen describes the program of the psychiatric service which includes supportive therapy, suppressive therapy, and dynamic psychotherapy—the last being more intensive and for a few patients. This program has industrial psychiatry aspects: "Certain problems are common to industrial medicine in any setting, such as sick leave, compensation claims, reemployment and the like." These matters are discussed briefly. This program offers an opportunity for mutual education among the various doctors involved and leads to a better understanding of emotional problems. —G.C.T.

Mental Attitudes and Safety. K. Menninger.* Menninger Quart. 13:1 (Summer) 1959. (*The Menninger Foundation, Topeka, Kan.)

Accidents rank third as a cause of death in the United States. Surprisingly, there is very little research on accidents, just as there is very little research into the factors responsible for suicide. The author asserts that less than 15% of accidents can be due to pure chance. Usual statements about the cause of accidents, such as that the person had drunk too much when driving a car, are really descriptive statements and they do not explain why, for example, a person is careless at a particular time. "There is a little murder and a little suicide dwelling in everybody's heart. Give them a powerful weapon like a car, inflame their inhibitions or irritations or frustrations, and diminish their suppressive control by means of alcohol or fatigue, and the murder or suicide may

get committed." The concept of the accident-prone individual has been widely held, and the author observes that all of us are accident-prone at various times. The question really is, what is our accident-proneness level at any particular time? Involved in accidents, then, are resentments, frustrations, and feelings of hostility and guilt. "The more mental health, the fewer accidents." —G.C.T.

Radiation Health

Health Hazards Associated with Rolling Normal and Enriched Uranium—Evaluation and Control. E. C. Hyatt.* Am. Indust. Hyg. A. J. 20:82 (April) 1959. (*Los Alamos Scientific Laboratory, University of California, Los Alamos, N. Mex.)

The allowable working level for both normal and enriched uranium is 70 disintegrations per minute per cubic meter. This represents 50 μ gm per cubic meter for normal uranium, which may be compared with cadmium or selenium, and 0.2 to 1.0 μ gm per cubic meter for enriched uranium, which may be compared with beryllium.

Air samples were taken during the heating, rolling, shearing, and storing of normal and enriched metallic uranium at the Los Alamos Scientific Laboratory and at two off-site locations. Exposures were shown to be principally to the inhalation of uranium oxide dust which was formed during the heating of billets and sheets. The exposures have usually been held to below 10% of permissible levels, as determined by air sampling and urine analysis. This has been accomplished by the use of appropriate control measures, mainly ventilation and the use of respirators when necessary. External radiation of biological importance from normal uranium is almost all beta at a surface rate of 250 mrep per hour. In the actual handling situation, experience shows that overexposures do not occur. The external radiation of enriched uranium is considerably less. —T.S.E.

The Concentration and Distribution of Radium in the Normal Human Skeleton. A. Walton,* R. Kologrivov, and J. L. Kulp. Health Physics 1:409 (March) 1959. (*National Physical Laboratory, Teddington, Middlesex, England)

Although relatively much information existed on the radium content of humans with toxic concentrations, data on the skeletal radium concentration of normal people had been limited. A large collection of bone samples, which had been gathered in connection with a strontium-90 project at the Lamont Geological Observatory, provided the material for such a radium study. Assay was done by ashing and subsequent radon counting. There was notable uniformity between different bone samples of the same individual, and between different individuals in the same area, and not great variation with geography. No significant correlation with age or sex was found. The worldwide average concentration was about 1×10^{-4} gm of radium per gram of ash, which is approximately 1/3000 of the maximum permissible concentration. —T.S.E.

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Metabolism of Radioactive Isotopes Which Deposit Mainly in the Skeleton. B. J. Stover.* Health Physics 1:373 (March) 1959. (*Radiobiological Laboratory, University of Utah, Salt Lake City)

More than 50 elements deposit in the skeleton to a significant degree. Of these, some are normal constituents of the organism, some behave chemically in a manner similar to normal elements, and some have no physiological analog. Not all radioactive versions of these elements are hazardous, simply because some are not available in sufficient quantities. The comparative toxicity of Ra^{226} and Pu^{239} has been studied extensively at the Radiobiology Laboratory of the University of Utah. The metabolism of radium is similar to that of calcium; therefore radium is deposited in areas of bone formation. Plutonium has no analog; it is concentrated on bone surfaces, and thus has higher concentrations in regions of high surface-to-weight ratio.

Beagle dogs were given single intravenous injections of either radium or plutonium. The concentrations of isotope were then followed in the blood, urine, and feces. Development of comparative quantitative metabolic information from this study will make possible refined estimation of human plutonium toxicity based on the known human effects of radium. —T.S.E.

Problems Presented by Radioactive Fallout: Statement by the General Advisory Committee, Committee to the U.S. Atomic Energy Commission.* J. W. Beams, M. Benedict, J. W. McRae, E. V. Murphree, K. S. Pitzer, J. C. Warner, R. E. Wilson, E. V. Wigher (Consultant), and W. C. Johnson, Chairman. Bull. Atomic Scientists 15:258 (June) 1959. (*Offices of the Atomic Energy Commission, Washington, D.C.)

This is a condensed review of a large mass of authoritative material regarding the problems created by fallout to date and that anticipated from weapons tests that have already been carried out. The Committee finds that the Atomic Energy Commission has released all significant fallout data to the public and to other agencies. The complexity of stratospheric circulation has made fallout less uniform than anticipated. The present best estimate is that about 100 pounds of strontium-90 has been injected into the entire stratosphere; 50% has fallen and 50% remains. To date the hazard from strontium-90 which has been found in food and water is less than that from radium normally present in the public drinking water supply in certain places in the United States. It is "pointed out that the amount of total body external radiation resulting from fallout to date, together with future fallout in any part of the world from previous weapon tests is less than 5 per cent as much as the average exposure from cosmic rays and other background radiation" or from medical x-ray exposures. The AEC must

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assure itself that it is conducting its own operations in a safe manner. Worldwide sampling of air, soil, foods, and water, and outstanding biological and medical research programs in the field of radiation hazards and protection have been operated to meet these needs. This is necessary to assure safety of its own operations and should be continued. "The relation between the public health authority and the AEC in its civilian activities should be analogous to that between the same authorities and most industry. In the opinion of the Committee, the public health agencies, both national and local, should gradually assume responsibilities for matters pertaining to the regulation of all radiation hazards affecting the public." The Committee felt that the level of effort the AEC has devoted to its research programs on radiation standards and protection in their broadest sense has been adequate. More understandable information should be given to the lay public about fallout. "It should be clearly explained to the public that weapons tests have been an essential part of our effort to prevent the occurrence of nuclear war." —W.D.N.

Clinical Toxicology

Toxicology of a Triaryl Phosphate Oil: III. Human Exposure in Operational Use Aboard Ship. H. D. Baldridge, D. J. Jenden, C. E. Knight, T. J. Preziosi, and J. R. Tureman. A.M.A. Arch. Indust. Health 20:258 (Sept.) 1959.

This report summarizes the findings of a clinical study of men who were associated with the use of a triaryl phosphate oil (TCP-1) in the hydraulic systems of two elevators aboard an aircraft carrier. In an earlier study of men engaged in the manufacture of TCP-1, no clinical findings attributable to TCP-1 were found; however, the plasma cholinesterase levels of the exposed personnel were significantly below the control levels.

In this study, no physical or neurologic abnormalities were encountered, and no significant systemic or neurologic symptoms were reported. Skin irritations that resulted from high environmental temperatures were not aggravated by exposure to TEP-1. Under the conditions of the test, TCP-1 had no adverse effects upon the plasma or red cell cholinesterase levels. Except for a statistically significant variation between subjects in the same group with regard to neutrophils and monocytes and significant variation in neutrophil counts done on different dates, no noticeable influence on the white and red cell counts were noted.

As a result of the findings observed in this study, it was concluded that toxic effects from exposure to TCP-1 are unlikely to occur under the conditions of operation that were in effect aboard the carrier. —L.H.M.

The Distribution and Retention of Chromium in Men and Animals. A. M. Baetjer,* C. Damron, and V. Budacz. A.M.A. Arch. Indust. Health 20: 136 (Aug.) 1959. (*615 N. Wolfe St., Baltimore 5)

Since epidemiological investigations have demonstrated a higher rate of bronchiogenic carcinoma among men employed in old chromate chemical manufacturing plants, studies were made to determine the distribution and retention of chromium in the body and the reactions of the tissues to chromium chemicals.

Lung tissues from 16 chromate workers (12 of whom definitely had lung cancer) were analyzed for chromium. Soluble and insoluble chromium was found even though the exposure had terminated many years previously. No correlation was found between the concentration of chromium (soluble or insoluble) in the lung tissues and the presence or absence of cancer. Bronchiogenic cancer tissue often contained little or no chromium. Likewise, little chromium was found in the bones of the chromate workers. In vivo and in vitro studies with guinea pig tissues showed that lung and liver tissues did not differ in their abilities to take up chromium. After water-soluble chromates were injected intratracheally in guinea pigs, only 15% of the dose was recovered from the lungs of the animals which were killed 10 minutes later, 20% was found in the blood, and a total of 5% was found in the liver, kidneys, and spleen. After 140 days, only small amounts of chromium were present in the lungs.

The mechanism by which soluble chromium is bound by the body tissues has not been elucidated. In the lung tissues of the chromate workers, the soluble chromium was bound firmly by cellular tissue and was insoluble in saline, acetone, and dilute base. The relative importance of hexavalent and trivalent chromium as factors in the production of lung cancer is discussed. —L.H.M.

Respiratory Disease of Mushroom Workers. L. S. Bringhurst,* R. N. Byrne, and J. Gershon-Cohen. J.A.M.A. 171:15 (Sept. 5) 1959. (*Department of Radiology, Chester County Hospital, West Chester, Pa.)

Sixteen cases of a respiratory syndrome were observed among migrant Puerto Rican workers who worked in the mushroom industry. Acute reactions in all patients occurred within a matter of hours after they had participated in the laying down or the removing of compost. Workers of local origin did not seem to be affected. Symptoms and signs which occurred with decreasing frequency were cough, rales, pain (epigastric, chest, and substernal), dyspnea, nausea, and vomiting, headache, chills, rapid weight loss, anorexia, sore throat, night sweats, malaise, diarrhea, hemoptysis, and epistaxsis. The acute phase usually subsided within one to six weeks after exposure had ceased. Recurrent attacks ("chronic" type of the disease) appeared to be mild. The roentgenologic findings consisted usually of diffuse infiltrations that were similar to those seen in azotemia. Oc-

casionally infiltrations were segmental, and, when seen, diffusely scattered minute seedings were seldom confluent and consolidative. Sputum examinations did not reveal a causative agent. Farmer's lung, thresher's lung, and silo-filler's disease have features in common with the syndrome of mushroom workers. The etiology has not been elucidated. Chest x-ray films from two cases are presented. —L.H.M.

Experimental Toxicology

Methoxsalen in Ultraviolet Carcinogenesis in the Mouse. A. C. Griffin.* J. Invest. Dermat. 32:367 (Feb.) 1959. (*Baylor University College of Medicine, Houston, Texas.)

When methoxsalen (8-methoxypsoralen) was injected intraperitoneally into mice (0.4 mg/mouse/day) one hour before exposure to the total spectrum of uviarc lamps (15 minutes/day for 100 days) more skin tumors and more erythemal damage developed than in the non-drug controls. Oral administration (0.5 gm/kg of Purina Laboratory Chow) gave less tumor incidence than the control and intraperitoneally injected groups; and less erythemal damage than the controls. Similar studies were conducted using 2537 Å lamps (20-60 minutes daily for 10 months). No difference was seen between tumor incidence in controls and the i.p.-injected groups, but no tumors appeared in the dietary group. With long-wave ultraviolet, no tumors developed in the controls but ear tumors developed in all i.p.-injected animals. Fluorescent light normally does not induce tumors but did in psoralen-injected animals. As yet there is no explanation for effects of psoralen on ultraviolet carcinogenesis. —R.E.E.

The Tumor-Promoting Action of Phenol and Related Compounds for Mouse Skin. R. K. Boutwell* and D. K. Bosch. Cancer Res. 19:413 (May) 1959. (*University of Wisconsin Medical School, Madison.)

Phenol, in benzene or acetone, was applied repeatedly to the skin of albino mice alone or after single applications of dimethylbenzanthracene (DMBA). Large numbers of papillomas appeared rapidly after treatment with DMBA followed by 10% phenol applications; carcinomas developed more slowly. Phenol alone elicited tumors also. The ability of 50 other phenolic compounds to promote tumors after DMBA application was studied. The response was dependent on the amount of phenol applied, the susceptibility of the mice, and the structure of the phenolic compound. —R.E.E.

The Dietary Toxicity of Glycerol Formal in the Rat. D. M. Sanderson.* J. Pharm. & Pharmacol. 11:416 (July) 1959. (*Fisons Pest Control, Ltd., Essex, Eng.)

Incorporated of up to 1,000 ppm glycerol formal in the diet of male rats (equivalent to 75-150 mg/kg/day by body weight) caused no significant toxic effects or metabolic changes. This amount is considered satisfactory for use as a solvent in dietary toxicity testing of solids insoluble in other less toxic solvents; inclusion of a solvent control is advised. Amounts greater than 1,000 ppm caused reduced weight gain, disturbance of drug metabolism, and increased susceptibility to infection. —R.E.E.

Effect of Chromium on Incidence of Lung Tumors in Mice and Rats. A. M. Baetjer,* J. F. Lowney, H. Steffee, and V. Budacz. A.M.A. Arch. Indust. Health 20:124 (Aug.) 1959. (*615 N. Wolfe St., Baltimore 5)

An experimental attempt to reproduce in the laboratory, conditions which in chromate chemical manufacturing plants are believed responsible for a high rate of bronchogenic carcinoma in workers was on the whole unsuccessful. Exposures of mice and rats to a mixed chromate dust (chromate roast) by inhalation and intratracheal and intrapleural injection did not result in bronchogenic carcinoma or in an increase in the incidence of benign lung tumors, although spontaneous tumors appeared at an earlier age in the dust-exposed mice than in the controls. Neither basic potassium zinc chromate nor barium chromate caused bronchogenic carcinoma or an increase in pulmonary adenomas when injected intratracheally or intravenously into mice. Intratracheal injection of zinc chromate produced epithelization of alveoli; this is being investigated further. Intravenous and intratracheal injection led to earlier appearance of adenomas. —R.E.E.

The Distribution and Retention of Chromium in Men and Animals. A. M. Baetjer, C. Damron, and V. Budacz. A.M.A. Arch. Indust. Health 20:136 (Aug.) 1959.

Analysis of lung tissue from men with industrial chromate exposures indicated no direct relationship between lung cancer and the concentration of soluble or insoluble Cr in this tissue. The lung tissue of man did not differ greatly from that of guinea pigs in ability to bind chromium, and in vitro studies in guinea pigs indicated that lung tissue had no greater affinity for chromium than liver tissue. Water-soluble Na_2CrO_4 or $K_2Cr_2O_7$, injected into animal tracheas, was largely removed from the lungs within a few days; small amounts were present after 140 days. Water-soluble $CrCl_3$ remained fixed for long periods of time at the site of injection. Water-soluble hexavalent and trivalent Cr was retained by tissues in saline-insoluble form. Lung slices bound chromic ions more readily than chromate or dichromate ions. The reaction with trivalent chromium seemed to be at the surface and to reach a fixed end-point. Part of the hexavalent chromium was reduced to the trivalent form in contact with lung slices. The slower but continuous uptake of hexavalent chromium might be due to a gradual penetration into the cells, suggesting that the

chromate anion plays a more important carcinogenic role than the trivalent cation. —R.E.E.

Noise and Hearing

The Measurement of Loudness. S. S. Stevens.* J. Acoust. Soc. America 27:815 (Sept.) 1955. (*Psycho-Acoustic Laboratory, Harvard University, Cambridge, Mass.)

The author reviews available data on subjective loudness. The objective was to reach a reasonable conclusion concerning the relation between loudness and the intensity of a sound from the assembled relevant data. Despite the various results obtained by workers, it is plain that people do make quantitative estimates of loudness and that it is not unreasonable to try to determine a loudness scale that will be representative of the typical listener. The empirical question is, "how do people describe sounds when we ask them to use a numerical language instead of adjectives"? The practical side of this problem had its origin in engineering acoustics. Soon after the decibel scale was applied to measuring sound intensity, it was found that equal steps on the decibel scale did not sound like equal steps. Frequently, a scale is needed that makes more sense to the ordinary person than do the numbers of the decibel scale. This need still persists.

Many attempts have been made to obtain direct estimates of the relation between loudness and intensity. Methods such as "magnitude estimation," method of constant stimuli, and method of adjustment have been employed in obtaining the data used in this review. The author discusses these methods and their limitations and also gives a brief description of the experiments producing the data. The principal data are given in four tables and certain relations among them are shown in three charts. The possible ways of testing the data are discussed. From his analyses, the author suggests that we accept the value 10.0 db as the intensity ratio corresponding to a loudness ratio of 2:1 for the 1000 cycle tone. The exponent of the power function then becomes $\log_{10}2$ and the formula for loudness becomes $L = kI^{0.3}$, where I refers to energy flux density and is assumed to be proportional to the square of the sound pressure. For sound pressure, p, the formula is $L = k^1p^{0.6}$. If I is measured in units equal to the reference level 10^{-16} watts per square centimeter and define L = 1 sone when I = 40 db, then k becomes 0.06 and $L = 0.06I^{0.3}$.

If the stimulus is measured as number of decibels N above the reference level, we can write log L = 0.03N —1:2. These relations hold for the 1000 cycle tone and may be different for other tones and especially for noise. These relations are also different from those currently used in computing "loudness of noise." Data from certain supplementary studies are presented and discussed. From the evaluation of all data the author devises a "possible engineering rule for measuring the loudness of noise." Possible formulae are given and discussed in terms of their limitations. Before drawing major conclusions, based on older methods of computing the "loudness" of noise, both engineers and medical specialists should become intimately familiar with the data pertaining to relation between loudness and strength of stimulus and their implications as presented by the author. —H.O.P.

Experiments on the Bone-Conduction Threshold in a Free Sound Field. C. W. Nixon* and H. E. von Gierke. J. Acoust. Soc. America 31:1121 (Aug.) 1959. (*Bio-Acoustics Branch, Aero Medical Laboratory, WADC, W-P AFB, Ohio)

The body-conduction threshold of hearing, when the head is immersed in a free sound field, has been computed approximately from data on the bone-conduction threshold when a small area of the forehead is excited directly. This work represents another approach to confirming the body-conduction hearing threshold for higher frequencies. If sound is conducted from the head surface to the inner ear by bone pathways when the ear canal is fully occluded, then the energy reaching the inner ear must be decreased if the head is covered by sound-absorbing material. Preliminary tests supported this reasoning and showed that a 2 to 3 in. layer of absorbent cotton, covering the head, provided enough absorption to give meaningful results for frequencies above 1,000 cps. All tests employed minimum-audible-field (MAF) hearing thresholds. All ear protectors were commercial items. Six test conditions were used: (1) open ear MAF threshold; (2) ear closed by ear plugs and ear muffs MAF threshold; (3) same as (2) except face covered with absorbent cotton; (4) same as (2) except back of head covered by absorbent cotton; (5) same as (2) except whole head (but not ear muffs) covered by absorbent cotton; (6) same as (5) except nostrils were clipped shut. The mean threshold shifts [condition (1) compared with condition (2)] introduced by five different ear plug-ear muff combinations were 30 db for 125 cps; 33 db for 250 cps; 37 db for 500 cps; 40 db for 1,000 cps; 42 db for 2,000 cps; 46 db for 3,000 cps; 52 db for 4,000 cps; 49 db for 6,000 cps; and 41 db for 8,000 cps. No single ear plug-ear muff combination, at the frequency for which it deviated most from the mean, deviated by more than about 5 db in the direction of less sound attenuation. Comparing these data with other published data indicates that for frequencies above 1,000 cps the thresholds obtained using the combination protectors represents the tissue-conduction thresholds for subjects in a free sound field. If this is so, covering the head with sound-absorbing material should raise the detection threshold—that is, require a higher sound pressure level about the head before the sound is heard. This was tested, using test conditions (3) through (6). In all cases, covering part or all of the head with cotton required a stronger sound field for

detection. For test conditions (5) and (6), an increase of 15 to 20 db in the level of the test frequency was required for frequencies of 2,000 cps and higher. These results suggest that a sound-absorbing covering for the head may provide increased protection for frequencies above 1,000 cps in special cases. —H.O.P.

Environmental Physiology

Salt-Deficiency Heat Exhaustion. C. S. Leithead,* L. A. Leithead, and F. O. Lee. Ann. Trop. Med. & Parasitol. 52:456 (Dec.) 1958. (*Department of Tropical Medicine, Liverpool School of Tropical Medicine, Liverpool, England)

Salt-deficiency heat exhaustion is a hazard to workers in hot industries and also to unacclimatized visitors to hot climates. Uncompensated loss of salt in sweat will deplete body reserves, leading to a reduction in extracellular fluid volume, which is the underlying cause of the clinical manifestations. Vomiting and anorexia, if present, increase negative salt balance still further. The authors report clinical and laboratory findings in 49 cases among oil tanker crews visiting the Persian Gulf in summer months.

Major symptoms are fatigue (100% of cases), giddiness (80%), anorexia, nausea, vomiting, and muscle cramps (60%), constipation (50%), headache (40%), diarrhea (30%), and syncope (10%). Oligemic shock was present in three cases. Elevated pulse rate and low pulse pressure were found in 25 others. Oral temperature tends to be subnormal (97°F average). Reduced blood sodium and chloride with elevated blood urea nitrogen and plasma proteins together with virtual absence of urinary sodium and chloride are the chief laboratory findings. Treatment requires bed rest in cool surroundings together with intake of salted fluids by mouth, or by vein if indicated by the presence of shock or vomiting. Treatment is continued until urinary chlorides exceed 3 gm/liter (as NaCl), up to five to seven days in severe cases. To prevent this illness in shipboard personnel in the Persian Gulf an intake of 20 to 30 gm of salt per day is recommended. —D.M.

On Physical Heat Regulation and the Sense of Temperature in Man. T. H. Benzinger.* Proc. Nat. Acad. Sc. 45:645 (April) 1959. (*Naval Medical Research Institute, Bethesda, Md.)

Heat loss from the skin to a warm environment is regulated by the anterior hypothalamus through autonomic control of sweating and cutaneous blood flow ("physical heat regulation"). Many physiologists regard the heat loss center of the hypothalamus as a relay station for afferent impulses arising in cutaneous thermoreceptors, changes in internal body temperature merely augmenting or depressing its excitability. Benzinger rejects this view and presents substantial evidence to indicate that hypothalamic temperature *per se* is the stimulus concerned in regulation of sweat rate and less exclusively of cutaneous blood flow. Afferents from cutaneous thermoreceptors appear to have no neural connection with the anterior hypothalamus although temperature sensations as such play an important role in body temperature control by modifying conscious behavior ("Pavlovian regulation"). These conclusions are based on experiments in the gradient calorimeter in which evaporative sweat rate was measured as heat loss from a human subject under steady state conditions at temperatures from +10 to +45°C and at three levels of metabolic activity. Rate of sweating when plotted against skin temperature showed no consistent pattern but did reveal a remarkably close linear relationship with internal temperature which was measured by a thermocouple on the tympanic membrane. The threshold for sweating lay between 36.80 and 37.00°C. For each 1/100°C rise above this threshold sweat rate increased by 6.2 grams per hour. Cutaneous blood flow measured as skin conductance bore a similar but less precise relationship to cranial temperature. Receptor cells in the hypothalamus are regarded by Benzinger as terminal sensory neurons, a temperature "eye" so to speak, whose threshold and response characteristics are specified above. —D.M.

Failure in Temperature Regulation During Progressive Dehydration. A. R. Hertzman* and I. D. Ferguson. WADC Technical Report 59-398, Aeromedical Laboratory, Wright Air Development Center, Wright-Patterson Air Force Base, Ohio, July, 1959. (*Department of Physiology, St. Louis University School of Medicine, St. Louis)

Fourteen healthy young medical students (mean values for height, weight, and surface area: 181 cm; 77.3 kg; 2.04 M^2) were exposed at rest and unclothed for 12 hours to a constant ambient temperature of 43.3°C (110°F) with vapor pressures from 20.7 to 33.5 mm Hg. Water and food were withheld, leading to progressive dehydration, which occurred at an average rate of 180 gm/M^2/hr. Oral temperature rose 0.9 degree (C) during the last seven hours. Sweat rate during exposure failed to increase, and cutaneous blood flow tended to decrease. This is in contrast to observations in well-hydrated subjects, in whom the authors previously reported an increase in sweat rate of 300 gm/M^2/hr. per 1 degree (C) rise in oral temperature (compare with Benzinger, 1959).

Failure of body temperature regulation during progressive dehydration is consistent with findings in previous studies by others which show that the threshold for sweating increases with rising osmotic pressure of tissue fluids. —D.M.

Acute Anhidrotic Heat Exhaustion. R. Bannister.* Lancet 2:313 (Sept. 12) 1959. (*Academic Unit, The National Hospital, Queens Square, London, W.C. 1, England)

Abstracts

Twelve patients with heat illness, differing in major aspects from recognized disorders caused by heat, were admitted to the British Army Hospital at Aden in September-October, 1958. The syndrome was characterized by a sudden decrease or cessation of sweating in young unacclimatized males during or immediately after exertion in severe heat. The mean morning effective temperature during this period was 93°F in the sun. Oral temperature on admission did not exceed 102°F. Thus the syndrome differed from heat hyperpyrexia and heat stroke, in which body temperature is 106°F or higher. Signs and symptoms were mild compared with those of heat stroke, consisting chiefly of exhaustion, drowsiness, and mental confusion.

There was moderate dehydration but no salt deficiency. Intercurrent infection was not noted. The skin was flushed hot and dry but was free of eruptions. Body temperature subsided at bed rest without recourse to special cooling; sweating returned to normal within a few hours. Thus the disease in these cases also differed from anhidrotic heat exhaustion of the type described by Ladell and others which is a sequel to severe prickly heat and requires weeks for return of normal sweating. The author suggests the term "acute anhidrotic heat exhaustion" for the cases observed at Aden. Recognition of this disorder is important to prevent its progress to heat stroke and to clarify etiologic factors which may be common to the two conditions. —D.M.

Industrial Hygiene

Some Hazards in the Manufacture and Use of Plastics. D. K. Harris.* Brit. J. Indust. Med. 16: 221 (July) 1959. (*From Imperial Chemical Industries, Ltd. [Plastics Division])

Initially the author is concerned with the incidence of dermatitis in British synthetic resin plants where formaldehyde is used and also with the toxicity of chemicals used in manufacturing polyvinyl chloride compounds. Figures are included which indicate that the incidence of dermatitis in the synthetic resin plants is decreasing. The hazards of polytetrafluoroethylene are detailed after mention is made of some hazards associated with the production of new polymers in the polythene group. Products that are formed when polytetrafluoroethylene is heated to very high temperatures may prove harmful when inhaled. The use of contaminated tobacco by men working with this material is among the commonest of ways by which they become exposed to the harmful effects of polytetrafluoroethylene. Harris points out, however, that although animal experiments have demonstrated that there is actual danger in heating polytetrafluoroethylene to extremes, it has not been possible thus far to establish precisely which of the by-products is hazardous, owing to the fact that differences are observed in the symptomatology of animals and humans exposed to this material.

The author gives a detailed description of the hazards in manufacturing copolymers of butadiene and observes that the toxicity of the monomers presents a hazard unless precautionary measures are taken when the latter are used in various processes.

In the interest of public health in general, it is of the utmost importance that the plastics used to package food and other consumer articles do not contaminate the products enclosed in them. Harris mentions certain procedures used to test the suitability of certain plastics for use as materials for packaging and describes in some detail several problems encountered in this regard. Later, the attention of the reader is drawn to the use of plastics in surgery. Harris observes that relatively little is known about the effects on body tissue of plastics used in surgery and he suggests that "the misuse of plastics in this field presents dangers that may not always be appreciated." —M.K.

Pulmonary Talcosis: A Clinical and Environmental Study. J. Messite,* G. Reddin, and M. Kleinfeld. A.M.A. Arch. Indust. Health 20:408 (Nov.) 1959. (*Division of Industrial Hygiene, New York State Department of Labor, 80 Centre Street, New York 13)

In 1940, an x-ray survey of 221 miners and millers employed in St. Lawrence County, New York, revealed pulmonary fibrosis in 32 workers. It was concluded that the fibrous type of talc to which the men were exposed was capable of causing a fibrogenic pneumoconiosis.

This paper presents medical and environmental data of a follow-up study, performed in 1958, of talc miners and millers in the same county. In addition, medical and environmental data obtained from talc mines and mills in two geographically separated regions of northern New York are compared and a discussion of the significant differences observed is presented. Procedures used in the study are described and a variety of tabulated data is included.

The study in 1958 showed that environmental controls of the talc hazards are vastly improved over those found in the study in 1940. This improvement is reflected in the medical findings of a very low incidence of pulmonary talcosis in the miners and a diminished severity and progression of the disease among the millers.

Statistical comparison of talc millers in the different geographical areas disclosed that the degrees of exposure to talc dust were similar and that the incidence and severity of pulmonary talcosis were of lesser magnitude where workers were exposed to a nonfibrous variety of talc.

The data in the paper indicate that both fibrous and non-fibrous talc can produce pulmonary disease but that the former is the more pathogenic. [AUTHORS' ABSTRACT]

About the Authors

IRVIN H. BLANK, Ph.D., is a product of the University of Cincinnati. From this institution he received the B.S. degree in 1924, the M.S. in 1927, and his doctoral degree in 1928. There followed six years of research in a variety of problems related to the conversion of animal skins to leather, in Boston and in Cincinnati. In 1934 he went to Harvard University as a Research Fellow in Botany, and in the following year moved into the basic research program in dermatology conducted by the faculty of Harvard Medical School at the Massachusetts General Hospital. Since 1955 he has been an Associate in Dermatology at Harvard and Associate Biochemist at "Massachusetts General."

DR. BLANK has produced 40 articles on dermatology, 4 on general microbiology, and 4 on leather chemistry and microbiology. He is co-author of one monograph on external therapy of the skin. His current fields of interest are basic principles of cutaneous topical therapy; water content of the stratum corneum; bacterial flora of the cutaneous surface; and percutaneous absorption. The last corresponds with the subject matter of Dr. Blank's article in this issue entitled "Percutaneous Absorption: Methods of Study and Factors Which Influence Absorption."

M. H. SAMITZ, M.D., M.Sc. (Med.), is senior author of the article "Extraction by Sweat of Chromium from Chrome Tanned Leathers." DR. SAMITZ received the M.D. degree from Temple University, Philadelphia, in 1933, and interned at Mount Sinai Hospital in that city. He was in specialty training in dermatology at the Graduate School of Medicine of the University of Pennsylvania during the years 1939-1942, and from that institution he received the M.Sc. (Med.) degree in 1945. After short periods at The Johns Hopkins University School of Hygiene and Public Health in 1942 he entered military service for two years. DR. SAMITZ was certified by the American Board of Dermatology and Syphilology in 1945. He then became Chief, Department of Dermatology, at the Graduate Hospital; and in 1949 became Assistant Professor of Dermatology at the University of Pennsylvania, in the Section of Industrial Dermatology. DR. SAMITZ is a member of various associations pertaining to his own specialty and is a member of the Industrial Medical Association.

SIDNEY GROSS, M.D., is associate author of the article, "Extraction by Sweat of Chromium from Chrome Tanned Leathers." He completed both his general college work and his medical education at the University of Pennsylvania. Following this, in 1952-1953, he served an internship at the Philadelphia General Hospital. He completed his residency training in dermatology at the University of Pennsylvania in 1958, where he continues as an instructor in dermatology.

DR. CHARLES D. CALNAN, of London, is author of the article in this issue on cement dermatitis. This presents views based upon extensive original work which the author has carried on for some time.

DR. CALNAN acquired the B.A. degree from the University of Cambridge with First Class Honors in 1939; and that University's M.A. and M.B.M.Chir. degrees in 1942. He became M.R.C.P., University of London, in 1950. His war assignment was as Specialist in Dermatology, Royal Army Medical Corps, during the years 1943-1947, three of which were served in India.

DR. CALNAN is Consultant Dermatologist to three London hospitals: The London Free Hospital, St. John's Hospital for Diseases of the Skin, and Hampstead General Hospital. He is Physician-in-Charge, Department of Industrial Dermatoses, Institute of Dermatology, in London. During part of 1959 he was in this country as a visiting research associate in the Department of Dermatology of the University of Pennsylvania.

CHARLES E. LEWIS, M.D., Sc.D., is co-author of the article in this issue, "The Ruster in Industry." He

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About the Authors

is plant physician at Texas Eastman Company in Longview.

DR. LEWIS' undergraduate education took place at the University of Kansas and the University of Kansas City, and led to a degree in chemical engineering. Thereafter he attended medical school at Harvard University, graduating with the M.D. degree in 1953. After an internship he served for two years as an internist with the U. S. Air Force in Southeast Asia. In 1956 DR. LEWIS entered graduate training in occupational medicine at the University of Cincinnati. As a part of this program he served an in-plant year at Eastman Kodak Company, Rochester, N. Y. He received the Sc.D. degree in industrial medicine from the University of Cincinnati in 1959. Since going to Longview, DR. LEWIS has joined the volunteer faculty of Baylor University College of Medicine, Houston, as a Clinical Assistant Professor of Epidemiology.

DR. LEWIS is the author of 11 articles in the field of industrial medicine and toxicology. He is a member of the Industrial Medical Association, serving on its Toxicology Committee, and is a member of American Industrial Hygiene Association and of Sigma Xi.

GEOFFREY A. HODGSON, M.D., of Cardiff, Wales, in this issue presents a substantial piece of original work in his article, "The Tolerance of the Skin to Lubricating and Cutting Oils." DR. HODGSON is Lecturer in Dermatology at Welsh National School of Medicine and has for some years been Dermatologist to the United Cardiff Hospitals. He is also Consultant Adviser in Dermatology, Wales Regional Hospital Board; Consultant Dermatologist, Ministry of Pensions Rockwood Hospital, Cardiff; and Member, Appeals Tribunal, Ministry of National Insurance. He is the author of numerous articles in the field of dermatology and of the chapter on "Hyperhydrosis" in the British Encyclopaedia of Medical Practice.

DR. HODGSON distinguished himself early as a scholar, with First Class Honours in Physiology, at Oxford University when he received the B.A. degree in 1932. He subsequently received additional degrees from Oxford: M.A.; B.M., B.Ch.; and D.M. (the last in 1940). The M.R.C.S., L.R.C.P. (Conjoint) was received at the conclusion of medical studies at St. Thomas's Hospital (London) in 1935. He became regimental medical officer in 1939, was promoted through successive grades as a dermatologist, and received the M.B.E. (Medical Division) for distinguished services in dermatology in N.W. Europe.

JAMES C. DRYE, M.D., is author of the first of a series of reviews on industrial trauma to be presented in this journal by members of the Department of Surgery, the University of Louisville. DR. DRYE'S introduction to this series, "Principles of Early Management," is a condensation of the current knowledge on the subject, and is based upon extensive research and experience with treatment of trauma both in civilian and military practice.

After receiving the M.D. degree from the University of Louisville in 1937, DR. DRYE spent four years as intern and surgery resident at Louisville hospitals. He entered active military service at Fort Knox in January, 1941, as a first lieutenant in the Army. Two years later he began active field service in Africa and Italy as a battalion surgeon, and then was attached to Second Auxiliary Surgical Group, with service in Italy, France, and Germany. The treatment of major injuries, and in particular the shock which attends such injuries, was a central interest of the surgeons who made up this Group.

DR. DRYE left military service as a major in 1945, and since 1948 has been a full time member of the faculty at the University of Louisville, giving special attention to the treatment of trauma at the Louisville General Hospital and to the cancer research program of the medical school. He holds the rank of Professor of Surgery.

DR. DRYE is certified by the American Board of Surgery, a member of several surgery associations, and of Sigma Xi.

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Dr. X. Y. Z. — age 33, available July 1960. Fellow in industrial medicine since July 1957—in-plant training. Is seeking a medical directorship at a salary of $15,000-$18,000. Prefers midwest.

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People and Events

► EDWARD V. HENSON, M.D., of South Charleston, West Virginia, was named Assistant Editor of the JOURNAL OF OCCUPATIONAL MEDICINE in a recent action of the officers and Board of Directors of the Industrial Medical Association. The appointment became effective with this issue of the journal.

DR. HENSON contributed substantially to the success of the journal in its first year. As a member of the first Editorial Board, he participated in the development of editorial policy, and made his share of appraisals of articles and reviews prior to acceptance for publication; he was the author of four topical reviews of the toxicology of families of organic chemicals; and since the middle of the year he has performed the editorial functions connected with the Abstracts Section. His specific responsibilities as Assistant Editor well be the continuing management of the Abstracts Section.

DR. HENSON graduated with a degree in chemistry from Pennsylvania State University in 1939, and received the M.D. degree from Jefferson Medical College in 1943. After an internship in Jefferson Hospital he served in the U.S. Army for three years, chiefly in the Pacific Theatre. He became a staff physician in the medical department of the Union Carbide Chemicals Company at South Charleston in 1947, and since 1950 has been the head of that department. He is a Fellow of the Industrial Medical Association, and is certified in occupational medicine by the American Board of Preventive Medicine.

Edward V. Henson, M.D.

► ROBERT C. COMBS, M.D., has accepted an appointment to the Editorial Board of the JOURNAL OF OCCUPATIONAL MEDICINE, filling the vacancy which resulted from the change of responsibilities of DR. E. V. HENSON.

DR. COMBS is a native of San Francisco and, except for four-and-one-half years spent in military service, continuously has been identified with that city. He received both his undergraduate and M.D. degrees from the University of California, the latter in 1939. His residency training in surgery was interrupted in 1941 by military service with the Army Ground Forces. He later completed his formal training in surgery, and he entered practice in San Francisco in 1947.

Besides an extensive background in surgery, much of which has dealt with the treatment of industrial injuries, DR. COMBS has also had substantial editorial experience. He was for two years editor of the Bulletin of the San Francisco Medical Society. Last year he was president of that Society.

DR. COMBS is now Assistant Clinical Professor of Surgery at the University of California. He is certified by the American Board of Surgery and is a member of the American College of Surgeons. As a Colonel in the California National Guard, he has responsibility for the 146th Evacuation Hospital, a 400-bed semi-mobile unit. A year ago DR. COMBS spent six weeks in

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People and Events

Europe as a guest of the German Government, making appraisals of hospitals and medical schools.

► DAVID JOE SMITH, M.D., has been appointed Plant Medical Director for the Edgar Thomson Works of United States Steel Corporation in Braddock, Pa. Since 1953 he has been Medical Director for L. S. Ayres & Company in Indianapolis.

DR. SMITH received his M.D. degree from Indiana University Medical School in 1942 and interned at the Methodist Hospital in Indianapolis. He served in the U.S. Army Medical Corps as a company commander in the 89th Infantry Division in the European theatre from 1943 to 1946.

Following military service, DR. SMITH completed three years' residency training in surgery at the Methodist Hospital, the last year as chief resident physician. During four years of private practice in surgery, he became associated with L. S. Ayres & Company as a part-time medical examiner, and assumed full responsibility for their medical program in 1953. Under his direction the medical department received the Occupational Health Institute's Certificate of Maintenance.

DR. SMITH is a member of the Industrial Medical Association and until his present appointment, was the IMA District Counselor for the State of Indiana.

► JAMES D. PEIRCE, M.D., has been appointed the Industrial Medical Association's District Counselor for the State of Indiana. The appointment was made by D. JOHN LAUER, M.D., IMA president. DR. PEIRCE is head of the Industrial Medicine and Safety Department for Eli Lilly and Company, Indianapolis.

► JAMES H. STERNER, M.D., has been elected President of the American College of Preventive Medicine. DR. STERNER is Medical Director for Eastman Kodak Company, Rochester, N.Y., and is co-chairman of the 1960 Industrial Health Conference. Other officers elected by the College include: JOHN D. PORTERFIELD III, M.D., President-Elect; ROY L. CLEERE, M.D., Vice-President for Public Health; THEODORE C. BEDWELL, Jr., M.D., Vice-President for Aviation Medicine; MELVIN N. NEWQUIST, M.D., Vice-President for Occupational Medicine.

► JACK M. PARTAIN, M.D., a member of the Texas Industrial Medical Association, has been elected to the position of President-Elect for the Texas Academy of General Practice. DR. PARTAIN is Company Physician for Carnation Creamery, Southwestern Bell Telephone and other companies in San Antonio.

► SEWARD E. MILLER, M.D., has been elected Chairman of the Occupational Health Section of the American Public Health Association. Other officers of the Section elected during the APHA annual meeting are: WILLIAM G. FREDRICK, Sc.D., Vice-Chairman; and MITCHELL R. ZAVON, M.D., Secretary. Elected to the Section Coun-

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cil were ELSTON L. BELKNAP, M.D., CLYDE M. BERRY, Ph.D., BERNARD E. CONLEY, Ph.D., LEWIS J. CRALLEY, Ph.D., CHARLES C. DILLS, M.S., and ROBERT H. FLINN, M.D.

► New York State Society of Industrial Medicine elected the following officers at its annual meeting on December 1: HARRY E. TEBROCK, M.D., President; THOMAS G. RIGNEY, M.D., Vice-President; GERALD J. FRIEDMAN, M.D., Secretary-Treasurer.

Elected to the Executive Committee were: ROBERT E. BRUBAKER, M.D., and ROBERT SIM, M.D., for three year terms; THOMAS H. DEELY, M.D., and JOSEPH PISANI, M.D., for one year terms.

► New England Industrial Medical Association at a meeting on November 4 unanimously voted for final incorporation under the Massachusetts law.

Officers elected at the meeting are as follows: FRANCIS LEBARON, M.D., President; HARRY MUSHLIN, M.D., Vice-President; and JAMES I. ROBERTS, M.D., Secretary. Elected to the Board of Directors were ROBERT CONDON, M.D., JOHN FINN, JR., M.D., EDWARD RUSSO, M.D., JOSEPH KAPLAN, M.D., DUNCAN MACDOUGALL, M.D., and GARFIELD DEFOE, M.D.

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